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OBSERVATIONS ON PULMONARY ARTERIAL PRESSURE AND PERIPHERAL VENOUS PRESSURE FOLLOWING ARTERIAL BLOOD LOSS

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THIS article is the first of a series concerned with pulmonary and circulatory changes resulting from simple peripheral arterial blood loss. Quite accidentally during experimental studies on pulmonary lymph flow in which controlled arterial bleeding was instituted, very consistent lung changes were observed[1] which were almost startling in their apparent import. Pulmonary edema and pulmonary hemorrhage were found to be produced at will by the simple removal of moderate to large quantities of blood from the femoral artery; the greater the blood volume loss, the greater the pulmonary damage (see Fig. 2).

A study of the physiologic factors involved in this pulmonary change seems very necessary at this particular time when great blood loss is such a common occurrence on the battlefields of the world. It was therefore decided to make an extensive study of a large group of dogs in which 25 per cent of the blood volume was to be removed (an equivalent to about 1,300 c.c. in a man of average weight), for it was thought that the loss of this quantity would simulate a battle casualty. A second series of experiments was performed in which a 10 per cent blood volume loss was used to compare with an operative hemorrhage of about 520 c.c. This group showed less severe but similar findings to the group in which the larger blood loss was sustained. A number of physiologic systems have been studied with a hope of correlating the picture of hemorrhage. It is of interest to observe that synchronous and uniform changes take place in many systems.

Following hemorrhage an immediate rise in pulmonary lymph flow occurs and is followed by a gradual fall to normal.[1] The per cent of pulmonary moisture is increased during the first 20 minutes, decreased below the normal during the next 20 minutes, again elevated during the third 20-minute period, and then gradually returns to the normal.[2] Both pulmonary arterial and peripheral venous pressures fall during the first 30 to 40 minutes after bleeding, rapidly

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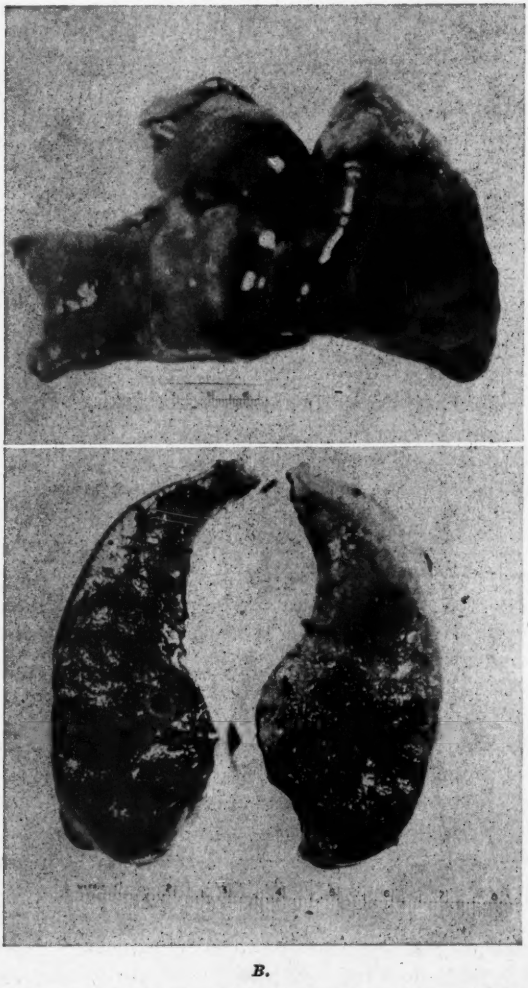
rise during the subsequent 30 minutes, and then stabilize at a level lower than normal. The respiratory effort increases in rate and depth during the 30 to 60 minutes following hemorrhage; there is evidence that the lungs are held in hyperinspiration for the whole of this period. The microscopic pulmonary picture is one of congestion during the first 10 to 20 minutes with intra-alveolar edema and hemorrhage, marked decrease in edema during the second 20-minute period with overdistention of the alveoli, congestion of the capillaries and marked edema during the third 20-minute period followed by a return to a normal picture at the end of four hours.[3]

A.



B.

Fig. 1.—*A*, Right lung of a dog in which a loss of 25 per cent of blood volume was sustained from the femoral artery four hours before sacrifice. Note dark mottled areas of hemorrhage throughout the lower lobe and the lower portions of the upper and middle lobes. *B*, Sections through right and left lower lobes showing extensive hemorrhage throughout the right lower, and in portions of the left lower, lobes.

It would seem that at the very time of acute hemorrhage or during the first few moments following acute arterial blood loss, important microscopic changes become evident. These are pulmonary hemorrhage and edema. A lapse of time of from four to five hours is required usually to make the hemor-

rhage recognizable in the gross appearance of the lung. Indeed it must be emphasized that these microscopic changes do not take place one or two hours after blood loss, but that they occur almost immediately at the time of the loss.

From a therapeutic standpoint, the stasis of the circulation within the lung resulting from pulmonary congestion is transient, and the condition clears in a matter of one and one-half hours if given an opportunity. This occurs in spite of the fact that the hemorrhage is not plainly seen by the naked eye until after the lapse of several hours. It would also appear that injudicious intravenous therapy, instituted at a time when there is pulmonary congestion and such marked increase in venous pressure, might aggravate the pathologic condition and lead to a fatal outcome from progressive pulmonary edema and

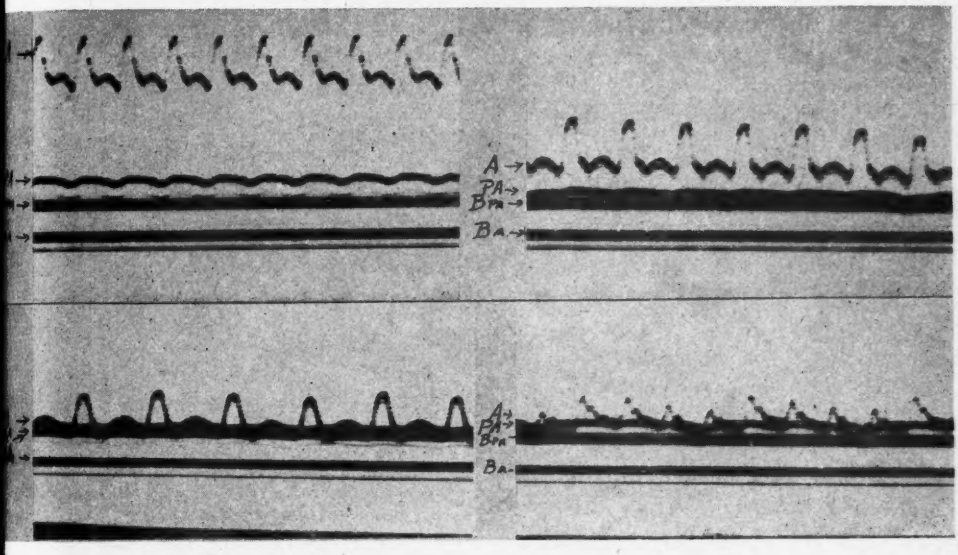


Fig. 2.—Arterial (carotid) and pulmonary arterial pressure curves recorded by Wiggers (optical) manometers. *A* shows normal pressures in the two arterial systems before hemorrhage (systemic arterial 120/80; pulmonary artery 30/15). In *B* a sharp drop in pressure in both systems has occurred following hemorrhage. In *C* there continues to be a drop in pressure in both systems. In curve *D*, the systemic arterial tension is low (approximately 80/50) and the pulmonary arterial tension has again risen (25/10 mm. Hg).

A, Arterial pressure; *PA*, pulmonary artery pressure; *Ba*, base line of *A* (zero); *BPA*, base line of *PA* (zero).

right heart failure. Studies have been made on the changes in the percentage of pulmonary moisture when acute hemorrhage is treated by various types of intravenous therapy which was started at the time of blood loss. Both venous pressures and pulmonary moisture percentages showed elevations over the expected rise which follows hemorrhage.[4]

A more complete discussion of the physiologic factors involved in the production of a pulmonary dam to normal circulation following hemorrhage will be presented.

METHOD

Young 35- to 40-pound heparinized* dogs under nembutal anesthesia† were used throughout the experimental work. Each dog was tied supinely on a

*In forty consecutive experiments with induced hemorrhage in which neither heparin nor intravenous therapy was instituted, the microscopic pulmonary changes were identical to the findings of those experiments in which heparin was used as an anticoagulant.

†In five consecutive experiments only local anesthesia with novocain was used. The results obtained were identical to those with nembutal. The anesthetic agent (nembutal) which was used in most of the work cannot, therefore, be considered an important factor in producing such pulmonary changes.

portable operating wooden frame and the femoral arteries and femoral veins, as well as the external jugular vein, were dissected free. The dogs and the frame were then placed on a fluoroscopic table where a catheter was inserted into the external jugular vein and passed by manipulation and fluoroscopic observation into the right main branch of the pulmonary artery by way of the superior vena cava, right auricle, and right ventricle, using the technique of Czebrinski and associates.[5] When this maneuver was satisfactorily accomplished the catheter was connected to a water manometer by means of a short rubber tubing, and mean pulmonary artery pressure impulses were transmitted to a moving kymograph by a cork carrying a fine piece of reed and crossbar. Using the same type of water manometer with cork and reed, pressure from the femoral vein was

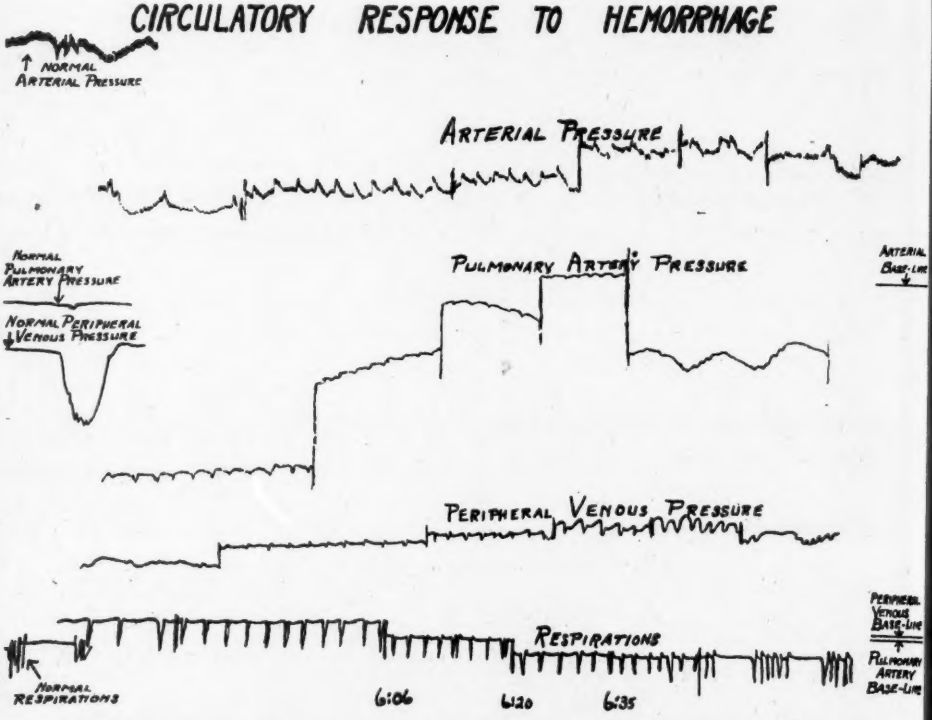


Fig. 3.—A photograph of an exact tracing of a twelve-inch strip from a nine-foot kymogram. Due to the delicacy of the line recording the pulmonary artery and peripheral venous pressures, a photograph of the kymogram was impossible to obtain.

This figure shows circulatory response 30 minutes after acute hemorrhage from the femoral artery (5:40 P.M.). The previous normal records are on the left side and the base lines are on the right. Note the gradual increase in the arterial blood pressure and the marked abrupt increase in pulmonary artery pressure which quickly reaches a peak and then recedes. Note also the rise in the entire respiratory tracing which preceded pulmonary artery increase and which is probably due to overdistention of the lungs following hemorrhage.

conducted to the kymograph through a T cannula and tubing for mean peripheral venous pressure readings. A standard type mercury manometer from a cannula in the femoral artery was used to record arterial blood pressure. Respirations were recorded by the usual type of spirometer. The zero base-line reading for each manometer was taken before and after each experiment, and adequate control readings were taken before bleeding was instituted. A short kymograph record was made every ten minutes during the experiment. Estimations of blood volume were made on a basis of one-thirteenth of the dog's body

weight, and when hemorrhage was to be instituted blood was allowed to flow freely from the femoral artery (note Fig. 3).

In one experiment, systolic and diastolic pulmonary arterial pressure and arterial blood pressure were recorded on a continuous photographic screen by means of Wiggers manometers attached to the cannulae (note Fig. 4).

RESULTS

Following the withdrawal of 20 per cent of the blood volume (5 per cent loss allowed for operative procedure) an immediate drop of pulmonary arterial pressure, systemic arterial pressure, and peripheral venous pressure occurred. During the subsequent 15-minute period a slight but perceptible rise in all pressures was noted. From 20 to 25 minutes following blood loss, the pulmonary arterial pressure increased abruptly and within the next 20 minutes it often reached its previous recorded normal level, occasionally exceeding that level. Examination of Fig. 3, which is a copy of a section of a kymogram, shows the abrupt and marked increase in pulmonary artery pressure which occurs for a short time after peripheral arterial blood loss.

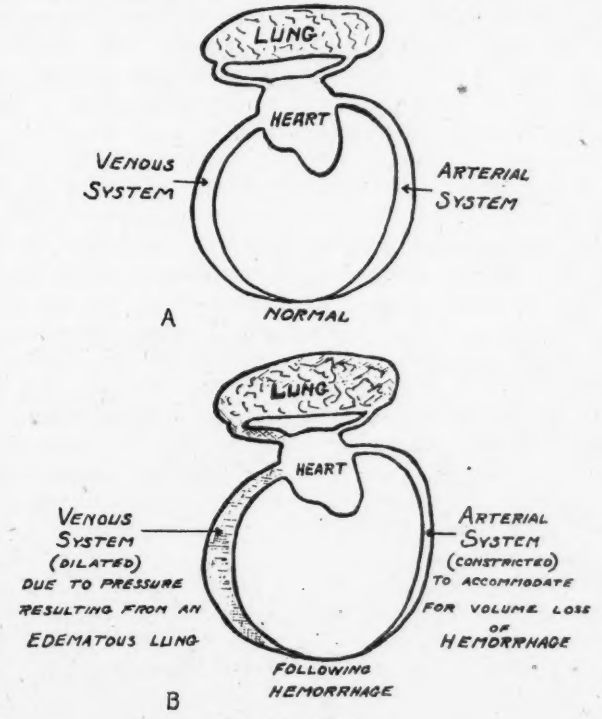


Fig. 4.—*A* and *B*.

The peripheral venous pressure began rising 10 to 20 minutes after the rise of pulmonary arterial tension. The venous pressure usually reached its previously recorded level 45 minutes after blood loss. Peripheral venous pressure increase has always shown this delay over the rise in pulmonary arterial pressure. It is probably necessary for this time to elapse before right ventricular dilatation occurs and for the venous pressure gradient to be affected.

The pulmonary arterial pressure and the peripheral venous pressure continued to be elevated for approximately 30 minutes. Thereafter, a gradual decline in both pressures was noted, and the tensions were stabilized at levels below the controls.

Arterial blood pressure was not observed to return to its normal level following a 25 per cent blood volume loss. As an example of a typical response, a pressure of 130 mm. Hg is present before hemorrhage, and a pressure of 90 mm. Hg occurs at the highest point after hemorrhage. In other words, the normal relationship between the arterial and venous systems was temporarily upset and the pressure in the latter was, comparatively speaking, much greater than that of the arterial system (note Fig. 3).

There was marked change in respiratory effort during and following blood loss. Respiratory rate was increased; there was an increase in the expiratory and inspiratory effort with evident forced expirations and inspirations of greater than normal depth. These changes of respiratory pattern continued for one or two hours following hemorrhage, after which there was a gradual return to normal.

DISCUSSION

The findings described were consistently observed in seven consecutive experiments in which pulmonary arterial pressure and peripheral venous pressure were recorded. Five other experiments were done in which peripheral venous pressures were recorded in combination with arterial and respiratory tracings. The same response was recorded as mentioned previously. It became apparent almost at once that the rise in pulmonary arterial pressure and in peripheral venous pressure following hemorrhage reflected either a profound disturbance in the pulmonary circulation or myocardial embarrassment.

It would seem possible that the pulmonary congestion resulting from systemic arterial blood loss is the result of increased permeability of the alveolar endothelium due to acute anoxia of the tissues. When the physiologic events are reconstructed, an acute anoxia is apparent for many reasons. Breathing is noted to be rapid, deep, and forced; air hunger has been produced and CO_2 concentration increased.[6] With arterial hemorrhage there is a true loss of blood volume and a decrease in available red corpuscles to carry oxygen. Heart rate increases in hemorrhage, but cardiac output actually decreases.[7] There is evidence that the circulation time is greater and that vasodilatation occurs. Besides these factors, there are certain experimental observations which should be mentioned which show that deep rapid forceful respirations and anoxia can produce increased pulmonary transudation.

In 1921 Graham,[8] using both the dog and the human lung suspended in a bell jar, showed that expiration will produce pleural effusion by the very squeezing effect of the respiratory effort, and that this effusion is increased if the respirations are exaggerated. Drinker and Warren,[9] in 1943, showed that both forced, labored breathing and reduced oxygen supply increase pulmonary transudates. They demonstrated marked increase in pulmonary lymph from the right pulmonary lymphatic duct of the dog when a greater intratracheal negative pressure was induced or when oxygen-poor mixtures of inspired air were used. In this latter experiment it was pointed out that anoxia which is so important for altering the permeability of the pulmonary capillaries toward increased leakage was the factor in increasing transudation and lymph flow.

Following these observations, cannulations of the right lymphatic duct were conducted in this laboratory; under controlled conditions it was found

that within 15 minutes of an acute hemorrhage there was a marked increase in pulmonary lymph flow.[1] It was felt that the two factors of forceful respirations and anoxia must be prominent in this change. In addition, microscopic sections of lungs from dogs killed 10 to 20 minutes after systemic arterial hemorrhage showed intra-alveolar edema and hemorrhage, as well as gross evidence of hemorrhage both on the pleural surfaces and throughout the parenchyma.[3] Also it has been shown by extensive desiccation and dehydration experiments that there is an immediate though transient increase in pulmonary moisture content within 10 minutes after a 25 per cent blood volume loss.[2]

Of significance in the present consideration is the observation of Landis[10] concerning the increased permeability of capillaries under conditions of oxygen want. Landis showed that immediately after a 3-minute period of oxygen lack, fluid filtered through the capillary walls of frogs' mesentery at approximately four times the normal rate. The increased permeability of the wall also permitted the passage of proteins, thus reducing the effective osmotic pressure of the plasma proteins to nearly one-half their normal value.

That there may be myocardial embarrassment to account for some of the pulmonary changes presented in these experiments is possible but not probable. Left ventricular insufficiency will produce massive pulmonary edema, and more or less transudation into the pulmonary alveoli occurs in instances of chronic passive congestion of the lung.[11] Pulmonary edema is frequently not sufficiently extensive to be clinically evident when histologically there is coagulated albuminous fluid in at least scattered alveoli of the lower lobes. It has been generally held that pulmonary edema in left ventricular failure is due to increased pressure in the capillaries transmitted from this failing ventricle. On the other hand, in the light of the rapid and profound pulmonary changes produced in these experiments, which seem to result from anoxic disturbance, one wonders whether in the pulmonary edema of left ventricular failure, the factor of anoxia may not be more important than is generally held.

It cannot be said unequivocally that in these experiments myocardial failure had no part in the pulmonary edema of arterial blood loss. However, the level of the arterial pressure following hemorrhage remained at a reasonably constant (lower than normal) level until the animal either recovered or died. When death occurred following hemorrhage it appeared to arise from a general circulatory collapse and respiratory failure. It seems possible, therefore, that cardiac failure was not primarily concerned in the disturbance of pulmonary circulatory dynamics observed here. It has been observed in the Starling heart-lung preparation[12] that pulmonary edema may suddenly develop and become marked long before the left heart shows any signs of failure.

It seems probable that the edema of the lungs produced in our experiments reduces vital capacity and that considerable acute compensatory overdistention of the lungs is produced. Evidence of considerable overdistention of the alveoli is noted in microscopic sections of dogs killed 20 to 45 minutes after arterial blood loss, and during this same period, following hemorrhage, there is often marked elevation of the respiratory curve on the kymograph, which signifies a lung held in hyperinspiration (note Fig. 3).

The data observed in these experiments are similar to those noted in experimental pulmonary embolism, in that the arterial pressure falls and venous pressure rises and that dyspnea is produced.[13]

The schematic drawings in Fig. 4 illustrate, in general, the effects of hemorrhage as observed in these experiments. Because operative and traumatic blood

loss is arterial blood loss, there is an immediate imbalance in the vascular bed. Volume has been lost from the arterial system. Arterial blood pressure falls due to decreased cardiac output and compensatory vasoconstriction. Venous pressure drops slightly for a short time, and the heart action increases in an attempt to equalize this unnatural state of affairs. It is improbable that any significant quantity of blood could be removed from the arterial system without affecting the equilibrium of the entire circuit and impairing the efficiency of the heart. As previously stated, cardiac output is markedly decreased following hemorrhage in spite of the increased rate. Thereafter the lung is waterlogged (see Fig. 3), and because of this impairment of normal circulation there is increased pressure within the venous system and a dilatation of the venous vascular bed. If, at this time, large quantities of intravenous fluid (saline, plasma, or blood) were administered, one can easily imagine that with the already dilated venous system, additional fluid may be impounded into the lungs, with serious consequences.

The observations here presented are consistent with those of the anatomic and clinical findings in "bomb blast lung" (and in many related explosive injuries), a pathologic condition brought about by instantaneous overdistention of the alveoli combined with direct trauma to the chest wall.[14] The most typical lesion of bomb blast injury is hemorrhage and edema of the lungs and there is clinical evidence of high venous pressures.[14] If by dog experimentation it could be shown that high venous pressures and pulmonary artery pressures are produced by blast, valuable application might be made to war injuries. Venesection within one hour of injury might be more vigorously stressed[15] and could conceivably be a lifesaving procedure, as in the acute pulmonary edema of congestive heart failure.

Hooker[16] has shown that *immediately* after experimental bomb blast there is a fall in venous pressure and that a hemorrhagic edematous state is found within the lung. In view of the pathologic findings described by him it seems possible that a mechanism may exist in bomb blast lung which is similar to that which we have described. It is suggested that if their observations had been carried over a longer period of time following the blast injury, changes in circulatory dynamics similar to those described here might have occurred.

SUMMARY

1. Pulmonary hemorrhage and edema result as an immediate reaction to acute arterial systemic blood loss.

2. Pulmonary congestion resulting from acute arterial blood loss produces a block to normal circulation and an increase of pressure within the peripheral venous system as well as within the pulmonary artery. This pressure is developed within 30 to 40 minutes of the blood loss and reaches its peak 50 to 60 minutes from the time of hemorrhage.

3. There is a gradual decline of these elevated venous pressures at a lower than normal level within one and one-quarter hours of the hemorrhage.

4. Intravenous administration during this short period of increased venous pressure has been shown experimentally to elevate the venous pressure additionally.[4]

5. The observations here presented are consistent with those of the anatomic and clinical findings of bomb blast lung and related conditions. Proof of this fact might make it desirable to try venesection as a lifesaving procedure, as in the pulmonary edema of congestive heart failure.

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CONGENITAL COARCTATION OF THE AORTA AND ITS SURGICAL TREATMENT

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IN EXPERIMENTAL studies on dogs in 1935-1936, one of us (C. C.) demonstrated that the flow of blood to all the organs could remain suspended for as long as twenty to twenty-five minutes without there being any subsequent signs of organic damage, provided an adequate flow to the brain was secured. This circulation to the brain was maintained by creating anastomoses between the carotid and jugular vessels on one side in the animal under operation with the corresponding vessels in a dog of the same size lying beside it. On the strength of this observation Crafoord, in certain patients with a patent ductus arteriosus, took the risk of placing clamp forceps on the aorta above and below the point of entry of the duct into this artery and of keeping them attached during the time necessary to divide the duct and suture the aorta. In one of the patients this part of the operation took no less than twenty-seven minutes to perform. In spite of this long period during which the aorta was shut off just below the point where the subclavian artery arises, no noticeable disturbances were subsequently observed in the patient's internal organs.

While performing this type of operation on other patients with a patent ductus arteriosus, Crafoord began to wonder whether it might not also be possible to treat congenital coarctation of the aortic isthmus by surgical means.

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As far as we have been able to ascertain from the literature, the only other investigator who has considered the possibility of relieving coarctation of the aortic isthmus by a surgical intervention is the American surgeon, Blalock. The latter, however, has attacked the problem in a more conjectural manner and from a different standpoint, having considered some form of anastomosing operation or plastic reconstruction to be the only way of improving the circulation peripheral to the stricture. He also states that no intervention to correct aortic stenosis has yet been performed on man.

In the light of the results obtained by Crafoord in the aforementioned experiments and of the clinical experiences gained from operations for patent ducti arteriosi, we considered that, in patients with congenital isthmic stenosis who already possessed a well-developed and satisfactorily functioning collateral system between the large arteries arising central to the coaretation and the arteries in the lower half of the body, we could venture to keep the aorta closed at a point immediately peripheral to the left subclavian artery for a period considerably longer than twenty-seven minutes without causing danger to the patient through cutting off the blood flow to organs in the part of the body supplied by arterial blood from arteries peripheral to the aortic stenosis.

In the discussion following a lecture on the ductus arteriosus read at a meeting of the Medical Society of Lund in 1944, Crafoord had already stated that he considered surgical treatment of aortic coarctation to be possible. The question was further discussed during the summer of 1944 with Nylin. By a strange coincidence it so happened that two patients with coarctation of the aorta were sent to the Sabbatsberg Hospital for an opinion at about the same time, the first to Nylin's department and the other directly to the surgical clinic. The first case had been interpreted as congenital vitium cordis of another kind, while in the other case the correct diagnosis had already been made. Both patients were first subjected to a thorough cardiologic examination in the department of cardiology.

The relatively bad prognosis in cases of aortic coarctation seemed to us to be sufficient justification for making an attempt at surgical treatment in these patients.

From the point of view of pathologic anatomy, an objection which might be raised against the performing of an operation in such cases is that, in post-mortem examinations, advanced changes of an arteriosclerotic nature have been demonstrated in the aorta and in the great vessels arising from it, situated central to the coarctation, and that in many cases it is precisely on account of these vascular lesions that death occurs. Pathologic changes of this type in the vessels must, however, be of secondary origin, consequent upon the heavy strain on the arterial walls caused by the constriction, and therefore, if operation could be undertaken at a sufficiently early stage, they would probably not be encountered.

Regarding the purely surgical aspect, the problem is simple. A condition of severe stricture is present in the aorta, which is a danger to the patient and should, therefore, be eliminated if the intervention can be done without undue risk.

Coarctation of the aorta is a fairly common abnormality, and our knowledge of this congenital deformity has accumulated in the last few decades. The anomaly has been known for nearly 200 years. Morgagni first paid attention to the abnormality in 1760, and the anatomist, Paris, made a thorough description of a case in 1789. According to Ask-Upmark, in 1897, Wadstein, from

Sweden, collected 103 cases from the literature. In her classical work on congenital cardiac disease, Maud Abbott found 142 cases of this abnormality in 1,000 cases of congenital cardiovascular defects.

From the surgical point of view, only the adult type is of special interest. The constriction of the aorta is localized just below or at the insertion of the ductus arteriosus. The narrowing may be of varying degrees, from a very slight one to complete obstruction of the aorta. The aorta is not usually dilated above the constriction, but not seldom the aorta proximal to the obstruction may be thin walled and hypoplastic. The descending aorta exhibits varying sizes in the individual cases. In some cases the descending aorta is of the same size as the upper part, but very often it is partly obliterated. The deformity is often associated with other cardiovascular anomalies, especially such as bicuspid aortic valves (20 per cent of the cases). Anomalies of the aortic arch are frequently found.

Generally the collateral circulation is greatly developed, because this is necessary to maintain the circulation below the level of the lesion. Generally the heart is enlarged and hypertrophy of the left ventricle is present. Atheromatous and degenerative changes in the aorta and the vessels of the head and neck are common. The enlargement of the heart and the degenerative changes in the vessels have been believed to be the result of the hypertension which generally accompanies this disease. Since Goldblatt introduced "the experimental hypertension" there has been much discussion in recent years as to the nature of the hypertension in coarctation. Paul White is of the opinion, in agreement with Steele and Cohn, that "hypertension is not to be ascribed to the effect directly but rather to a secondary effect, quite probably to the diminution of the renal blood flow below the constriction, which in turn causes a generalized vasoconstriction reflexly or by the production of the chemical mediator called hypertensin or angiotonin." In a later paper Steele devoted much attention to this explanation of hypertension in coarctation. Rytand is of the same opinion as Paul White.

Coarctation of the aorta in its classical form is easily recognized. The most important sign is the increased blood pressure in the upper part of the body. We may, however, find some cases of coarctation with normal blood pressure in the arms. Every young person with hypertension should be examined at first to find out if he has a coarctation, and if he has no pulsations in the popliteal artery or a lower blood pressure in his legs than in his arms, then it should be realized that there may be coarctation. Often, but not in every case, collateral pathways form tortuous and dilated arteries, which can be seen, and which can be palpated over the back, especially in the interscapular region, and even on the lateral part of the chest in the axillae. In most of the cases the dilated tortuous intercostal arteries cause erosions or notchings of the ribs, which are seen in the roentgen picture of the chest and, if present, they are a sign in coarctation. In rare cases no erosion is to be found. A systolic murmur is often heard over the precordium, especially over the base of the heart, and transmitted to the back, especially in the interscapular region and loudest to the left. There may be a diastolic murmur of aortic incompetence. By means of a new roentgenologic diagnostic method employed by Robb and Steinberg, in some cases the heart and the great vessels have been visualized by a radiopaque substance, diodrast, and the coarctated aorta has been shown on the roentgen picture. In one of Nylin's early cases in which the patient was not operated upon, Lysholm detected the coarctation without

any diodrast injection. In general the average age at death in Abbott's series was 32 years, with extremes of 3 and 92 years. The other causes of death were as follows: congestive heart failure, sudden death of the heart (rupture of the aorta and rupture of the heart), cerebral haemorrhage, and bacterical endocarditis. Regarding therapy, Paul White, in his last textbook, wrote as follows: "There is no special therapy for congenital coarctation of the aorta, but the subject should be protected as much as possible from physical strain and infections." To this statement may be added today the beneficial result of the operative treatment (the Crafoord operation), which is illustrated by Cases 1 and 2.

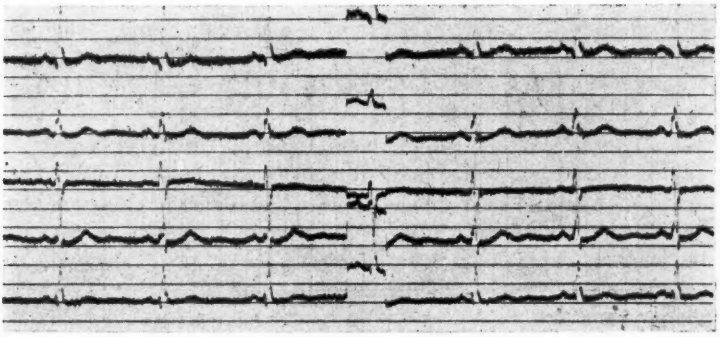


Fig. 1.—Electrocardiogram, Case 1.

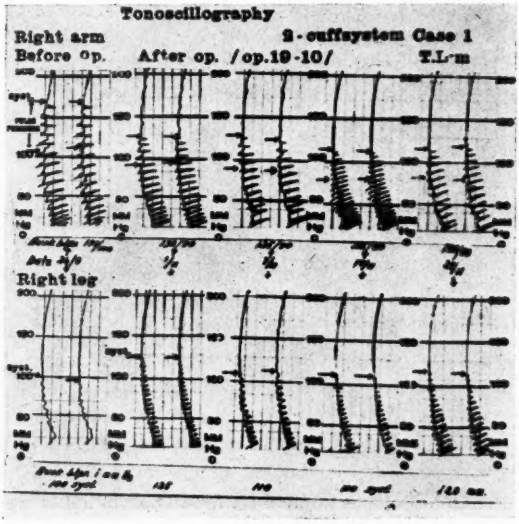


Fig. 2.—Tonoscillograms taken before and after operation (Case 1). In the arms (the upper curve) there are high blood pressure and good oscillations. In the legs (the lower curve) the pulsations are very small and the blood pressure low.

With the 2-cuff system in use here the systolic pressure lies at the point where the oscillations stop (marked by arrows). After operation the systolic pressure decreases in the arms and increases in the legs.

CASE REPORTS

CASE 1 (795/44).—T. L., a 12-year-old boy, was sent to Nylin for diagnosis of suspected ductus arteriosus Botalli persistens. The boy had been thin and delicate as a child. When he commenced school, he began to feel weakness, but played and ran about like other boys. Since 1942 he had had high systole blood pressure varying between 140 and 190 mm. Hg.

Physical Examination.—Examination revealed a slender pale boy. The heart was not enlarged. The apex beat was within the left midclavicular line. Over the whole precordium auscultation revealed a loud blowing systolic murmur loudest in the second intercostal space to the left of the sternum. Blood pressure in the arms was 170/105, in the legs 100/70.

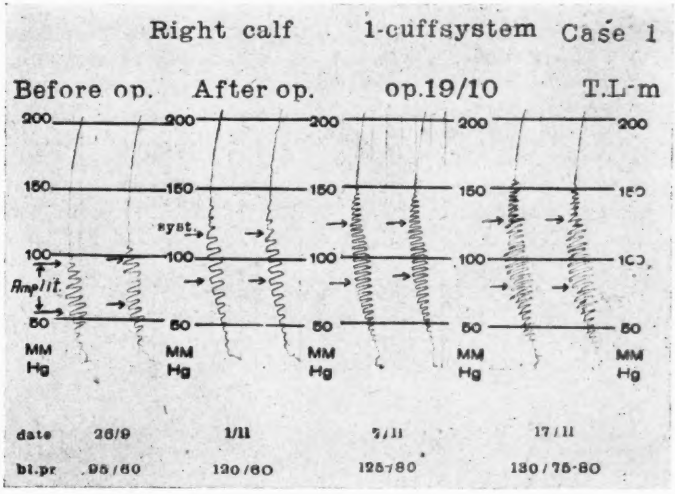


Fig. 3.—Tonoscillograms from the right calf of the patient (Case 1), before and after operation 1-cuff system. Both the systolic and the diastolic blood pressure increase after operation (marked by arrows).

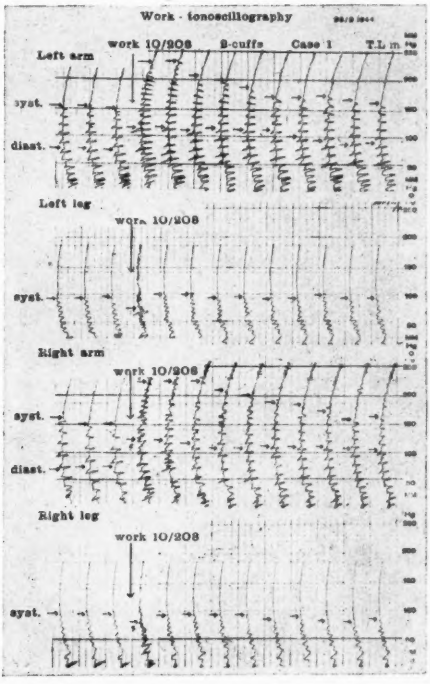


Fig. 4.—Case 1, work-tonoscillograms from both arms and legs taken before operation. (Hard work, ten rounds in the Nylin staircase at the rate of 208 steps per minute). From the legs there is pathologic response to work as has been shown by Ejrup (Nord. med. 23: 1770, 1944. Sv. för. för. inv. med. förh. 30: 1498, 1945.) We find the same type of response to work in the legs of normal patients as in the arms of our patients.

In the legs no pulsations were palpable, not even in the popliteal area. Collaterals: Under the left clavicle a strong tortuous pulsating artery was felt but nothing was palpated to the right. A strongly pulsating artery was felt also in the left axilla. Intercostal vessels were dilated and easily palpable under the third rib on the left side. Venous pressure was 5 cm., circulation time (decholin): first sensation after 6 seconds, maximum of sensation 9 seconds, disappearance of the bitter taste after 16 seconds.

Electrocardiographic Examination (Fig. 1).—There was regular heart action of 85 per minute; P-R interval was 0.12, QRS 0.08, TI and TII were upright. There was a small upright T in the third lead; T in the anterior and posterior chest leads was upright; left axis deviation.

Oscillography With the Ejrup Equipment.—

Before operation.—(Figs. 2, 3): In the arms, high blood pressure and good oscillations were found. In the legs, however, the pulsations were very small and the blood pressure low (below 100 mm. Hg).

After operation (Figs. 2, 3): Both the systolic and the diastolic blood pressure increased in the legs and decreased in the arms.

Work-tonoscillography Ad Modum Ejrup Before Operation (Fig. 4).—After hard work, (10/208, ten rounds in the Nylin staircase at a rate of 208 steps per minute) there was distinct increase of the blood pressure in the arms but no increase of the blood pressure and no increased pulsations in the legs. (After operation this examination has not yet been done.)

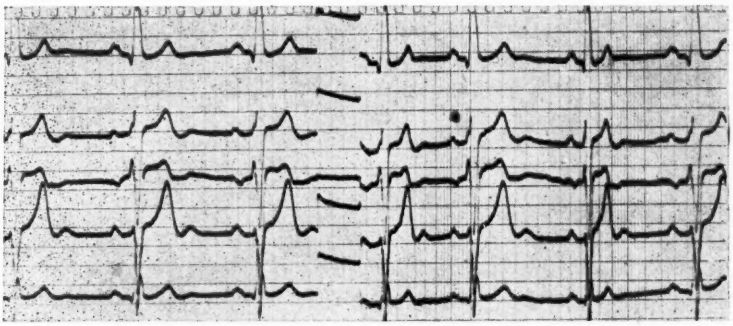


Fig. 5.—Electrocardiogram, Case 2.

Case 2 (809/44).—K. E. G., a 27-year-old farmer, was sent to Crafoord on a diagnosis of coarctation of the aorta. Until November, 1943, he had been in good health and had managed the heavy work as a farmer without difficulties. In November he became heavy headed, dizzy, and feeble. Hard work tired him. He had no pains but had a feeling of constriction in the heart region. Heart disease was diagnosed in March, 1944. Sometime afterward, high blood pressure was found. In August, 1944, the diagnosis of coarctation of the aorta was confirmed. Blood pressure, 210/110, was lower in his legs than in his arms. Electrocardiograms showed left axis deviation.

Physical Examination.—Examination revealed a rather stout-built man with a vivid complexion. The heart was enlarged. The apex beat was probably palpable in the fourth intercostal space. Over the whole, precordium auscultation revealed a systolic murmur loudest in the second intercostal space close to the sternum. The second sound was strongly accentuated in the same place and also over the apex region. Blood pressure in the arms was 180/100, in the legs less than 100 mm. Hg. In the arms good pulsations of the arteries were palpable; in the legs no pulsations were palpable or audible by auscultation. There were no signs of any collaterals palpable on the chest wall. Venous pressure was 10.5 cm. Circulation time (decholin): First sensation after 19 seconds, maximum of sensation after 27 seconds, disappearance of the bitter taste after 38 seconds.

Electrocardiographic Examination, (Fig. 5).—There was regular heart action of 60 per minute; P-R interval was 0.18, QRS 0.13, TI, TII, and TIII upright. T in the anterior and posterior chest leads was upright. There was left axis deviation. S-T was elevated in the second and third leads, depressed in the anterior lead. Ventricular complexes were notched.

Phonocardiographic Examination (Fig. 6).—There was a systolic murmur over the sternum on a level with the second intercostal space.

Oscillographic Examination With the Ejrup Equipment.—

Before operation (Fig. 7): In the arms high blood pressure and good oscillations were found. In the legs, however, the pulsations were very small and the blood pressure low (below 100 mm. Hg).

After operation (Fig. 7): Both the systolic and the diastolic blood pressure increased in the legs and decreased in the arms.

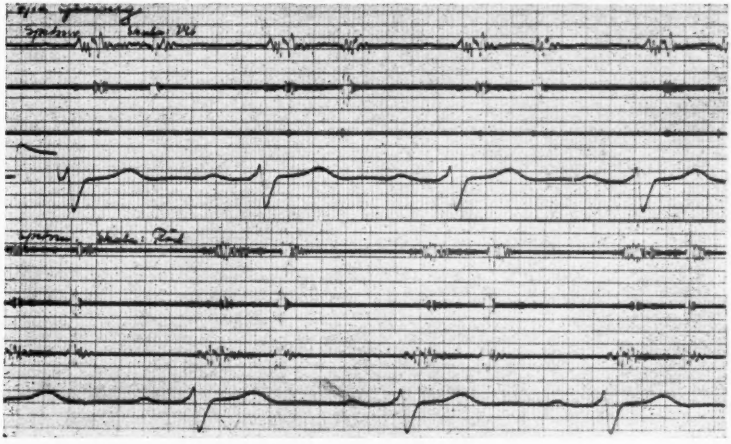


Fig. 6.—Phonocardiogram, Case 2.

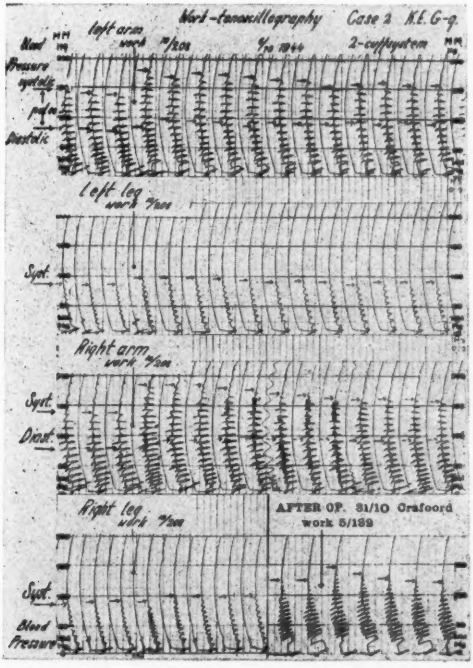


Fig. 7.—Case 2, Tonoscillograms and work-tonoscillograms before and after operation. Before operation we find the same curves as found in Case 1. Four and one-half months after operation new work-tonoscillograms have been taken and now the response to work is normal from the right leg. (Work after operation only five rounds in the Nylin staircase at a rate of 132 steps per minute).

Fig. 8.—Stenosis at the insertion of the ligament of Botalli. Collateral circulation between the dilated subclavian arteries and the descending aorta. (From Love, W. S., Jr., and Holms, J. H.: Am. Heart J. 17: 630, 1939.)

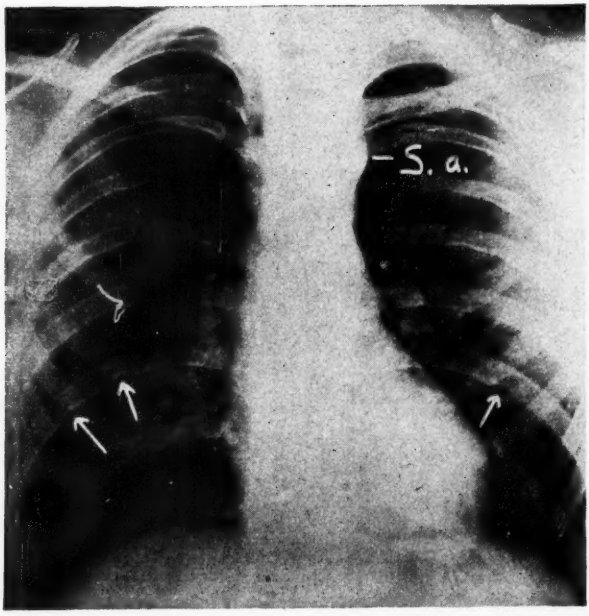
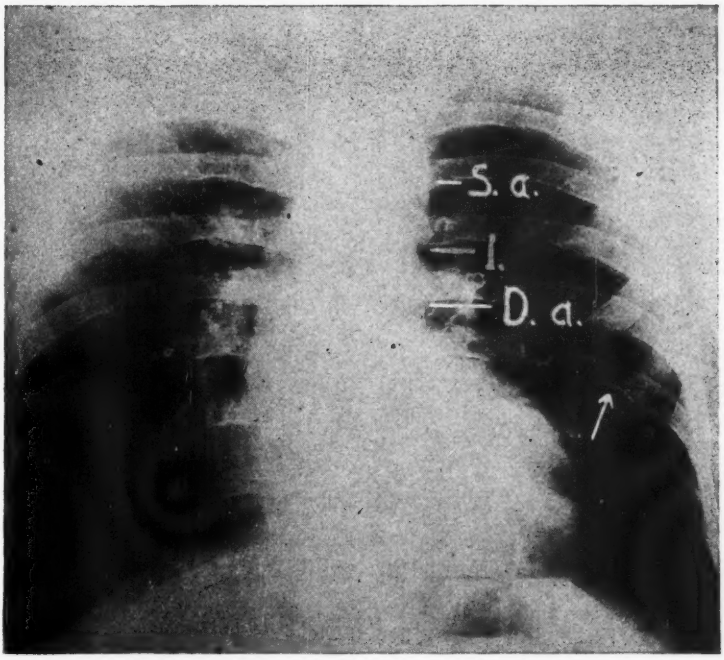


Fig. 9.—Case L. superior mediastinum widened. Convex outline to left of dilated left subclavian artery (*S.a.*), easily mistaken for aortic knuckle and rib defects. Cardiac volume before operation 450 ml., cardiac volume per square meter body surface 380 ml. Four months after operation, cardiac volume 375 ml., cardiac volume per square meter body surface 320 ml. (From Gladnikoff, H.: Acta radiol. 1946.)



A.

B.

Fig. 10 *A* and *B*.—Case 2. Heart enlargement, most strongly pronounced in left chamber. Ascending aorta shows increased curve on the right and increased amplitude in pulsations. Aortic knot cannot be demarcated, nor can aortic arch in lateral view. Left subclavian artery (*S.a.*) is easily mistaken for aortic knot on account of shortened aorta. Descending aorta (*D.a.*) and indentation (*I*) between left subclavian artery and aorta; rib defects. (From Gladnikoff, H.: Acta radiol. 1946.)

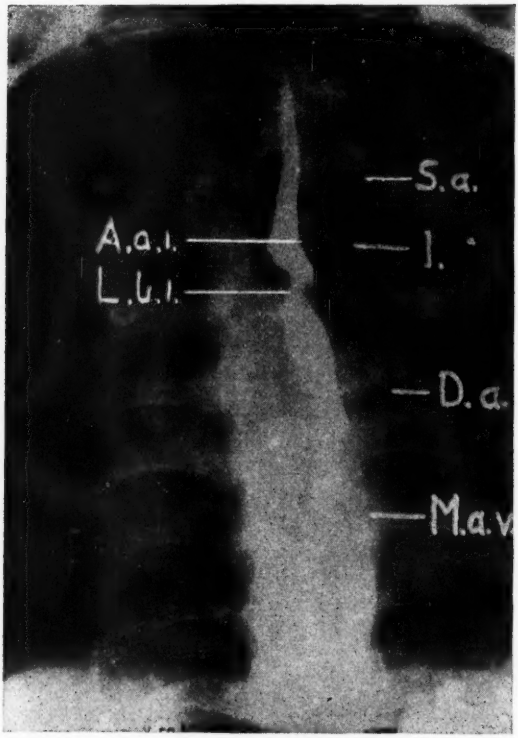
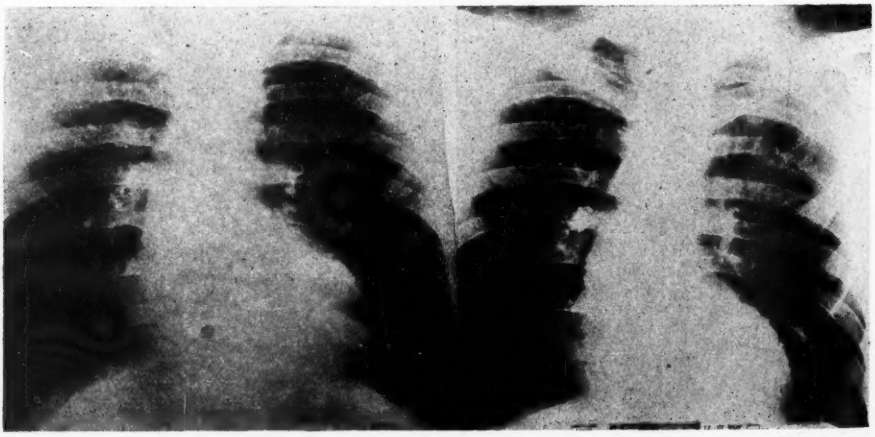


Fig. 11.—Superior mediastinum widened, left outline formed by dilated, left subclavian artery (*S.a.*). Indentation (*I.*) in posterior mediastinum where left subclavian artery leaves aorta; aorta shortened. Medial to aorta the coarsened minor azygos vein (*M.a.v.*) forms an outline, aortic arch impression (*A.a.i.*) on esophagus, left bronchus impression (*L.b.i.*) on esophagus. *D.a.*, Descending aorta. (From Gladnikoff, H.: Acta radiol. 1946.)



A. *B.*

Fig. 12.—Case 2. *A*, Before operation is cardiac volume 1120 ml., cardiac volume per square meter body surface, 620 ml.

B, Four months after operation is cardiac volume 880 ml., cardiac volume per square meter body surface, 465 ml. (From Gladnikoff, H.: Acta radiol. 1946.)

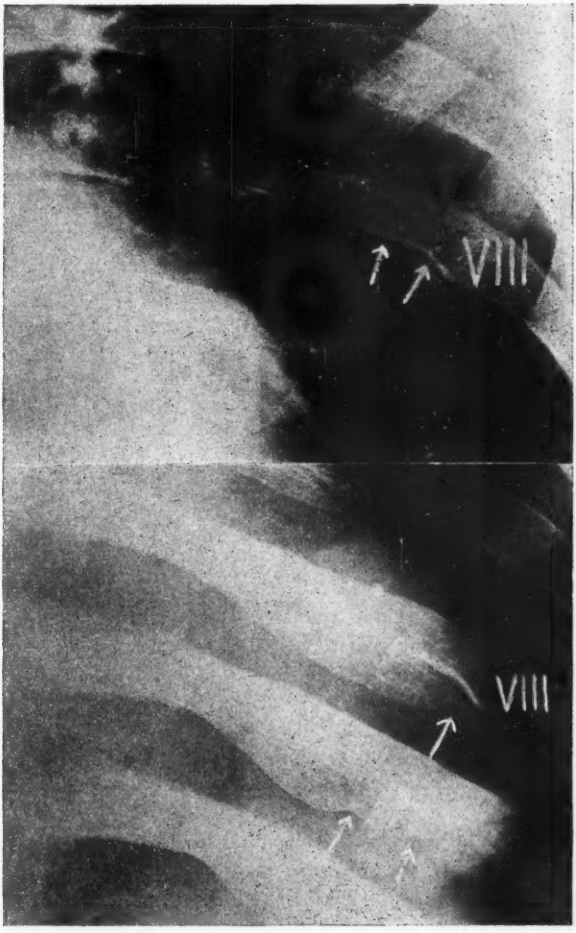
Work-tonoscillography Ad Modum Ejrup (Fig. 7).—

Before operation: After hard work (10/208) there was distinct increase of the blood pressure in the arms but no increase of the blood pressure and no increased pulsations in the legs.

After operation (Fig. 7): After slight work (only five rounds in the Nylin staircase at a rate of 132 steps per minute) the blood pressure increase in the legs as in normal cases. (Note the difference in the hardness of work before and after operation.) (See Figs. 8, 9, 10, 11, and 12.)

Roentgenographically, there are described in cases of coarctation of the aorta changes in the ribs, the heart, and various parts of the thoracic aorta as well as in the mediastinum.

A.



B.

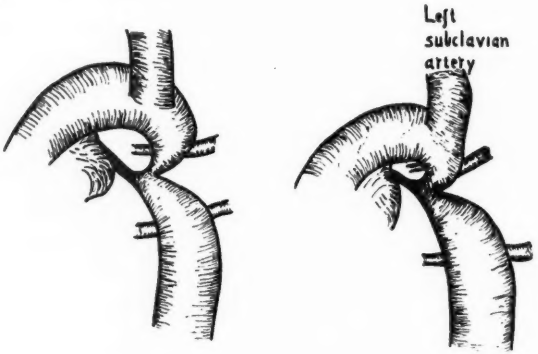
Fig. 13.—Case 2. Ribs: Costal sulci widened in several places. Bone is so thin here that structure cannot be observed on radiograms made with hard roentgen rays (*A*) but appears when rays of soft quality and a suitable diaphragm are used (*B*). (From Gladnikoff, H.: Acta radiol. 1946.)

The changes in the *ribs* (Railsback and Dock, Roesler, and Fray) include a widening of the costal sulci and defects in parts of the lower edges of the ribs (Fig. 13).

The heart is hypertonic; the left ventricle is most often enlarged and the ascending aorta usually shows an increased curve. The *shadow of the aortic knuckle* is curiously enough *absent* or very small (Assman, Schatzki and Hallerman, Kerley). The aortic arch is often impossible to outline even in the oblique view (Fray).

Gladnikoff of the roentgenological department of Sabbatsberg Hospital, who has examined our patients, considers that the operative finding will explain the impossibility or reduced possibility to observe parts of the aorta. These are drawn ventrally and medially by the ligament of Botalli, so that an extra V-shaped curve is formed (Fig. 14), with parts of the aorta entirely dislocated into the mediastinum. In the other parts of the aorta this extra curve causes a tautness, which must also dislocate these parts medially toward the mediastinum. The aorta will therefore be surrounded to a lesser extent than is normally the case by an air-conveying tissue and the possibility for observance will be diminished in the same degree.

The widened shadow of the superior mediastinum is strongly pulsating and is hence caused by arteries. The operation showed that the shadow to the left in the roentgen picture was formed by the considerably widened left subclavian artery.



A. *B.*

Fig. 14.—*A*, Case 1; *B*, Case 2.

In the posterior mediastinum approximately on a level with the fifth vertebra, there is an indentation (Figs. 10 and 11) of barely one centimeter's depth (Wolke). The operations showed that it was formed cranially by the outline of the left subclavian artery and caudally by the aorta. Near the place where the subclavian artery leaves the aorta its outline is convex laterally and has therefore been interpreted earlier as the aortic knot by several authors. (The roentgen findings will be more extensively described in a separate article by Gladnikoff which is to be published in Acta Radiologica.)

In both patients, the operation was planned as a trial thoracotomy, in order to ascertain whether the aorta wall was in a condition to permit circular suture of the vessel after resection of the stricture. If the aorta wall showed signs of morbidity or if, after mobilization, it seemed unlikely that the aorta could be made sufficiently mobile to allow coaptation of the resected edges without tension, after the removal of the stricture, we did not intend to continue the operation but simply to leave the stenosis as it was.

The operation was performed in both instances by Crafoord, the first Oct. 19, 1944, and the second Oct. 31, 1944, through the enlarged incision for

thoracotomy already described in his Thesis of 1938, the fifth rib being removed in both patients. Intratracheal insufflation anesthesia with an airtight tube was used for both operations, and in both cases, also, controlled respiration produced by the spiropulsator with cyclopropane-nitrous oxide as the narcotic agent was secured.

In both patients, the aorta showed conical narrowing from the origin of the subclavian artery, and the conical constriction between the coarctation and the point of departure of the subclavian artery was much longer in the first case (a small boy), than in the second. This feature may be seen from the accompanying drawings (Fig. 14).

In both, this part of the aorta seemed, as it were, to be drawn forward and in toward the median line, causing the segment narrowing off toward the coaretation to take a bend, with the concavity pointing medially. From the point of greatest constriction the aorta then widened out rapidly as it arched off backward and laterally, and within approximately 2 cm. of its length it had regained its normal diameter. A strange feature was that, peripheral to the stenosis, the aorta was in both patients of larger caliber than the actual aortic arch at the origins of the great vessels. It had been expected that, owing to the hypotonia on the peripheral side of it, the aortic lumen would be much smaller than it was on the central side. In both patients the lateral course of the aortic arch continued in an almost direct line upward toward the lateral contour of the first ascending part of the widely dilated left subclavian artery. Inspection and palpation of the aortic arch proved that there were powerful pulsations of its wall the entire way down to the stenosis. Below the constriction the dilated aorta was not moving, and no pulsations could be palpated.

The parietal pleura was divided lengthwise along the lateral segment of the aorta from the origin of the subclavian artery down to a point about 5 cm. below the stricture. Within the whole of this area the aorta was explored and its entire circumference isolated. This procedure presented no great difficulties except with regard to the area of greatest constriction. This segment was short, not more than 4 to 5 mm. in length, in both patients. The difficulty experienced in exploring the narrowest part was due to the fact that it was embedded in hard, fibrous tissue.

This fibrous tissue passed over into a cord of connective tissue not quite as thick as as a slate pencil, which could be readily isolated. Its commencement was on the medial posterior aspect where the stricture was most pronounced, and after a little more than 1 cm. it passed into the wall of the pulmonary artery exactly at the point where this divides into left and right branches. The recurrent nerve, after leaving the vagus, hooked around this fibrous structure before passing upward in a cranial direction toward the neck. The cord must, therefore, have been the remains of the ductus arteriosus. The intercostal vessels arising from the aorta on either side of the stricture were also explored. These vessels were greatly dilated, and slightly tortuous, both above and below the constricted segment. Clamp forceps were then attached to the aorta just below the point of origin of the subclavian artery, as well as at a point as far away as possible from the first pair of aortic intercostal arteries. Small clamp forceps were then placed on all the branches arising from the aorta between the two forceps on the main trunk of the aorta. Resection of the aorta, taking in the most constricted segment, was then carried out (for specimens, see Figs. 15 and 16). On the peripheral side, the incision through the aorta was made in a plane at right angles to the long axis of the aorta, 2 mm. proximal to the point

of origin of the first pair of intercostal arteries. Centrally, the plane of incision was placed at an angle of about 45 degrees in relation to the long axis of the aorta, owing to the fact that the lumen here was narrower than on the peripheral aspect, and about 1 cm. away from the area of greatest constriction. In this way, two lumina with circumferences to fit one another were obtained. A typical Carrel aorta suture was then made. First, three interrupted sutures were placed at equal distances from each other, and then running silk sutures were sewn between the interrupted sutures, each stitch being drawn up separately to secure adequate tautness (Fig. 17).

When the forceps were removed it was found that an ideal artery suture with no leaks had been achieved. In neither of the patients was there any difficulty in coapting the two lumina of the aorta and producing a suture without tension.

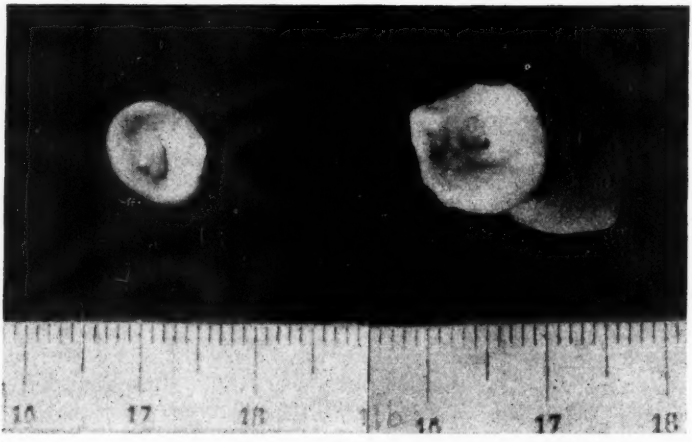


Fig. 15.

Fig. 16.

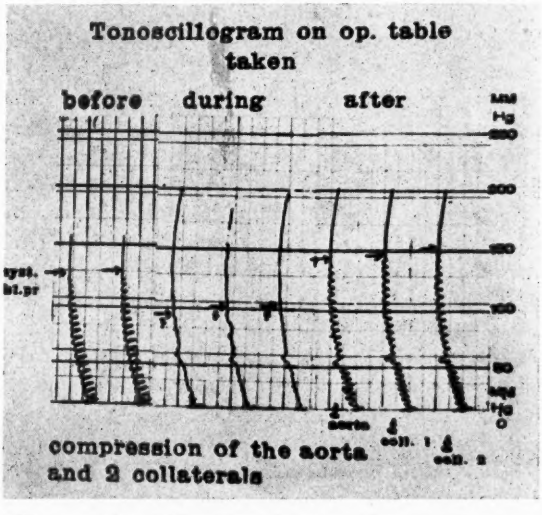


Fig. 17.—Oscillograms taken during the operation (Case 1). The two cuffs are placed on the right leg. While the aorta is cut off there are small pulsations in the legs. The blood pressure increased a little when the large collaterals which were clamped during the aorta resection were reopened. Note the fact that the pulsations are no bigger after than before operation.

In both patients the wound healed by primary intention. A sterile pleural exudate arose, as it usually does after our thoracotomies. It was treated by aspiration once or twice.

In the first patient there was a temperature elevation at the end of the second and the beginning of the third week, due to an acute upper respiratory tract infection of a nosocomial nature. The infection rapidly subsided and the temperature was never more than 38° C. In the other patient congestion of the left lung of bronchopneumonic type developed and the temperature rose. After treatment with a sulfa compound the congestion subsided and the temperature returned to normal.

In March, 1945, both patients were re-examined. Both were in excellent health. The boy had started school and the farmer worked a little at home. Oscillograms from the boy, showed blood pressure in the left arm 130/80 mm. Hg. and in the left leg 150/80 mm. Hg. In the second case (the farmer), oscillograms showed blood pressure in the left arm 145/90, in the left leg 170/90 mm. Hg.

It is remarkable that in both cases normal conditions re-established themselves regarding the blood pressure, so that in both cases the blood pressure in the legs was a little higher than in the arms and that no hypertension is now present. This postoperative effect is of considerable interest and is in favor of the mechanical theory of hypertension in coarctation.

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THE PROXIMAL EXTENSION OF CARCINOMA OF THE LUNG IN THE BRONCHIAL WALL

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CARCINOMA of the lung was until recently classified as one of the "hopeless" diseases even in its early stages. When the diagnosis of intrathoracic carcinoma was made, the physician's thoughts immediately turned to sedation and alleviation of pain to make the last weeks of the patient's life easier. Fortunately, this attitude has undergone some modification in the past fifteen years. The prognosis in carcinoma of the lung is still exceedingly grave but there is some basis for hope through the judicious use of pneumonectomy and lobectomy in many cases in which the diagnosis is made early.

The pathology of lung tumors has been very thoroughly studied by previous investigators some of whom have referred to the direct extension of the carcinoma along the bronchial wall. None, however, have measured the exact distance which the malignant cells may extend directly from the primary lesion, or the various layers of the bronchial wall in which this may occur. This is a study of the proximal extension of carcinoma along the various coats of the bronchial wall.

Cabot[1] reported a case in which a primary bronchiogenic squamous-cell epithelioma projected into the bronchus and grew along the bronchial tree, which he said rarely if ever occurs in the case of a metastatic tumor.

In 1940, Rabinovitch, Hochberg, and Lederer[2] noted that carcinoma arising from the epithelium which lines the bronchi is usually large and bulky. It advances along the bronchial wall after invading it and leads eventually to ulceration, obstruction, secondary infection, and even perforation. Carcinoma that originates in the mucous glands, however, is chiefly limited to the bronchial wall and usually produces infiltration and thickening of the wall which may ultimately cause stenosis of the bronchus. Halpert[3] also referred to the infiltration of the bronchial wall by the carcinoma and noted that the tumor may involve the surrounding tissue by direct invasion.

Geschickter and Denison[4] divided carcinomas of the lung into two types. The most common type is the bronchial or epidermoid carcinoma. They also referred to this type of hilar carcinoma and said that histologically it is composed of epidermoid cells which resemble those lining the walls of the large bronchi. The second type is the diffusely growing adenocarcinoma. "Microscopically the tumor has an adenomatous structure resembling the terminal ramification of the bronchioles and it may contain a mucoid secretion." This division of carcinoma of the lung into only two histologic types is not only the most convenient but also the most logical one when considered embryologically according to Geschickter and Denison.

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Bronchiogenic carcinomas are usually found in the region of the hilus of the lung. Fried[5] found that the right bronchus was more often involved than the left. He concluded that this was probably because the right bronchus is shorter, wider, and more vertical in direction than is the left bronchus. This permits the easier penetration by foreign bodies which leads to irritation and inflammation and to the ultimate development of a malignant change. In a study of thirty-seven cases, Rosedale and McKay[6] found the tumor present in the right lung in twenty cases and in the left lung in sixteen cases. In one case the site of the tumor was not recorded. Halpert stated that, "The most usual site of primary growth of carcinoma of the lung is at or near the hilus. The location is usually the point at which the stem bronchus divides or at which the secondary bronchi are given off."

Anderson and others[7] also found that the hilar type of carcinoma accounts for 90 per cent or more of all carcinomas of the lung. He found the site of origin to be in a main bronchus, commonly near the bifurcation. Usually the carcinomas take the form of a papillary growth or they may be diffuse and cause narrowing of the lumen by occlusion. Anderson concluded that only rarely does the growth spread by way of the mucosal surface. In only seven of a series of twenty-three cases reported by Mattick and Burke[8] was the lesion peripheral; in the remaining sixteen cases the lesion was central. The majority of tumors reported by Rosedale were situated in the first order or larger bronchi near the hilus. "In twenty-two cases the tumor was located about a first order bronchus, in three cases about a second order bronchus, and in one case about a third order bronchus."

In 1931, Verbrugghen[9] measured the extension of carcinoma of the stomach in and through the gastric wall. His study included fifty surgical specimens of gastric carcinoma. He found that the favorite plane for lateral extension in the gastric wall is the submucosa. In eleven cases the lesion had extended in the mucosa and in eight it had involved the serosa. From this study Verbrugghen concluded that, "The amount of extension from the edge of a gastric carcinoma varies according to the degree of differentiation (MacCarty) and the grading (Broders). In the most diffuse carcinomas of the stomach, the growth has no visible edge, and a proper point for resection cannot be indicated; in all other types of gastric carcinoma a distance of at least 4 cm. of healthy gastric wall should be allowed from the edge of the ulcer along the lesser curvature toward the cardia, in order that the resection may include the whole of the primary focus."

New and Fletcher[10] conducted a similar study on carcinoma of the larynx. One hundred specimens obtained at laryngectomy were studied. This survey revealed that in carcinomas grade 1 there was no extension, in carcinomas grade 2 there was an extension of 5 mm., in carcinomas grade 3 an extension of 5.9 mm., and in carcinomas grade 4 an extension of 5.6 mm. The explanation for the fact that carcinomas grade 4 did not extend further locally may be their tendency to invade the lymphatic channels early. New and Fletcher concluded, as did Verbrugghen, that the grade of the malignancy has a definite relation to the extension beyond the apparent limits of the tumor.

In 1942, Conway and Broders[11] reported a study of submucous extension of squamous-cell epithelioma of the urinary bladder. This study included more than 400 specimens of tumor of the bladder removed surgically and 150 specimens removed at necropsy. Sections were taken at specified distances from four sides of the tumor and examined microscopically for evidence of

extension of the carcinoma. Forty specimens when measured in the fixed, contracted state presented a margin of 1 cm. or more of tissue apparently free of tumor on from one to four sides of primary tumor. Conway and Broders noted the same decrease in percentage of extension in tumors grade 4, as did New and Fletcher.

MATERIAL

This paper is based on fifty-five cases of carcinoma of the lung in which the surgical specimens obtained by lobectomy or pneumonectomy were studied microscopically to determine the proximal extension of the malignant lesion along the bronchial wall. These cases were observed at the Mayo Clinic in a period of nine years and five months, namely, from Jan. 1, 1935, to May 31, 1944, inclusive. At present, lobectomy and pneumonectomy are being performed far more frequently than they were in the period covered by this study; therefore, the number of cases in this series is small. Of the fifty-five cases studied, only thirty were used in the final survey. The others were rejected either because the specimens had been mutilated by previous investigation or because the lesion obviously did not involve a bronchus. This investigation sought only to determine the proximal extension of the carcinoma beyond the gross limits of the tumor. The distal extension was not investigated because the purpose of this study was to determine how far proximal from the gross limits of the lesion the surgeon should sever the bronchus.

METHOD OF PROCEDURE

The lesion was located in the fixed tissue and a section including the proximal border of the gross lesion and the entire thickness of the bronchial wall extending proximally to the incision was cut. This block of tissue was then mounted in paraffin, cut longitudinally, and stained with hematoxylin and eosin. In the study of the slides the gross limits of the tumor were again determined macroscopically. A line was then drawn from this point vertically through the bronchial wall. Any malignant cells found proximal to this line were considered to be an extension of the primary gross lesion.

The tissue was then examined microscopically to determine and measure the proximal extension, if any, of the carcinoma. The carcinomas were graded from 1 to 4 by the method of Broders.[12]

The layers of the bronchus were divided by the method of Jordon[13] into the tunica mucosa, the tela submucosa, the cartilaginous coat or adventitia, and the outer fibrous coat. The tunica mucosa consisting of the ciliated epithelium lining the bronchus is the first layer. The second layer, the tela submucosa, is made up of connective tissue and mucous glands and lies between the mucosa and the cartilage. The cartilaginous coat consists of the irregular plates of cartilage found in the bronchial wall. The outer fibrous coat consists chiefly of loose, irregular strands of connective tissue.

RESULTS

The following types of tumor were encountered in the thirty cases: a squamous-cell epithelioma in sixteen, or 53 per cent; an adenocarcinoma in six, or 20 per cent; an adenoma in seven, or 23 per cent, and an indeterminate but rapidly growing type of carcinoma in one, or 3 per cent, of the cases respectively (Table I). This is in agreement with the established concept that more than one-half of all malignant tumors of the lung are squamous-cell epitheliomas.

TABLE I. SUMMARY OF DATA ON ENTIRE 30 CASES STUDIED

AGE (YR.)	LUNG	SEX	TYPE OF TUMOR	GRADE	EXTENSION
31	Left	Female	Adenoma	--	
42	Right	Female	Adenoma	--	
33	Left	Male	Adenoma	--	
42	Left	Female	Adenoma	--	
30	Right	Male	Adenoma	--	
31	Right	Male	Adenoma	--	
39	Left	Female	Adenoma	--	
50	Right	Male	Undifferentiated carcinoma	3	
47	Right	Male	Adenocarcinoma	3	
42	Left	Female	Adenocarcinoma	2	
38	Right	Male	Adenocarcinoma	4	
57	Left	Male	Adenocarcinoma	3	+
60	Right	Female	Adenocarcinoma	3	+
57	Right	Male	Adenocarcinoma	3	+
66	Right	Male	Squamous-cell epithelioma	3	
51	Left	Male	Squamous-cell epithelioma	3	
57	Left	Male	Squamous-cell epithelioma	2	
48	Left	Male	Squamous-cell epithelioma	2	
44	Left	Male	Squamous-cell epithelioma	3	
51	Left	Male	Squamous-cell epithelioma	3	
60	Right	Male	Squamous-cell epithelioma	3	+
51	Right	Male	Squamous-cell epithelioma	3	+
59	Right	Male	Squamous-cell epithelioma	3	+
59	Left	Male	Squamous-cell epithelioma	2	+
53	Left	Male	Squamous-cell epithelioma	2	+
54	Left	Male	Squamous-cell epithelioma	3	+
39	Left	Male	Squamous-cell epithelioma	2	+
57	Right	Male	Squamous-cell epithelioma	3	+
55	Left	Male	Squamous-cell epithelioma	3	+
49	Right	Male	Squamous-cell epithelioma	3	+

In thirteen, or 43 per cent, of the thirty cases, the lesion had extended proximally and had involved at least one of the coats of the bronchial wall (Table II). The distance that the tumor had extended in the thirteen cases and the coats of the bronchial wall in which the extension had occurred are shown in Table II.

In ten of the thirteen cases in which extension had occurred, the tumor was a squamous-cell epithelioma, grade 2 or 3. The minimal extension in these cases was 0.3 mm. and the maximal extension was 15 mm. In each of these two instances, the lesion had extended in the outer fibrous coat. In the latter instance, the lesion apparently had extended well beyond the point at which the involved bronchus was severed. In one case, a squamous-cell epithelioma had extended in the mucosa (Fig. 1) and in another case it had extended in the submucosa. In each of these two cases, the distance that the tumor had extended was 5 mm. In three of the ten cases, the tumor apparently had extended beyond the point at which the bronchus was severed. In two of these three cases, the extension had occurred in the outer fibrous coat (Figs. 2, 3, and 4); in the remaining case, it had occurred in the submucosa (Table II).

In two of the ten cases of squamous-cell epithelioma, the extension had occurred only in the mucosa. In both of these cases, the mucosa appeared normal on gross examination and the extension was discovered by microscopic examination.

Extension had occurred in three of the six cases of adenocarcinoma. Table II shows the distance that the tumors had extended and the coats of the bronchial wall in which the extension had occurred. The greatest extension observed in the three cases was 2 cm., which was by far the longest distance that a tumor had extended in any of the thirteen cases in which extension was observed. This is

Fig. 1. Fig. 2.

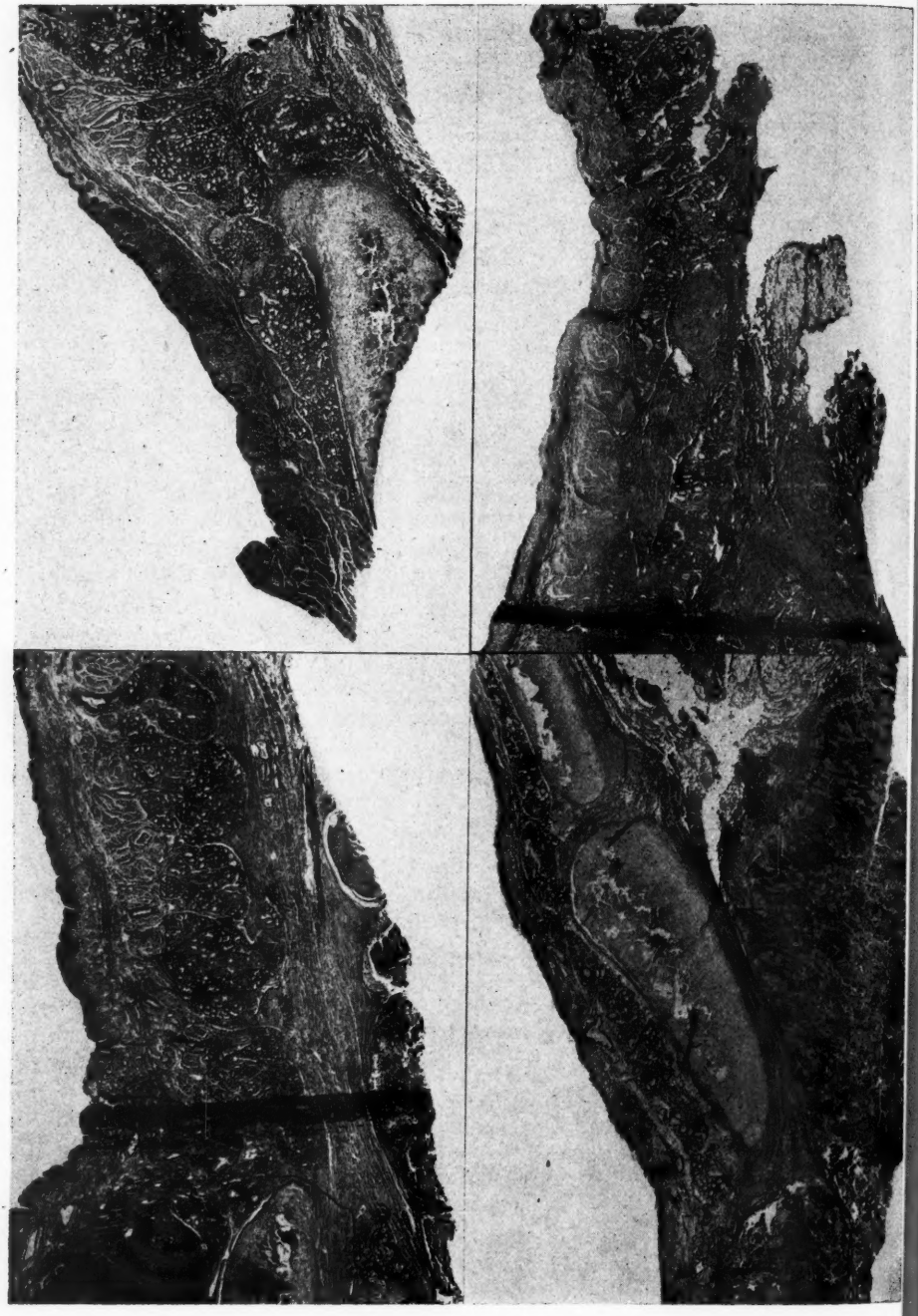

Fig. 3. Fig. 4.

Fig. 1.—Squamous-cell epithelioma with proximal extension into mucosal layer of bronchus for 5 mm. (×10½).

Fig. 2.—Proximal extension of squamous-cell epithelioma into outer fibrous coat of bronchus for 0.4 mm. (×10½).

Fig. 3.—Proximal extension of squamous-cell epithelioma into outer fibrous coat of bronchus for 1.3 mm. (×10½).

Fig. 4.—Proximal extension of squamous-cell epithelioma into outer fibrous coat of bronchus for 1.5 cm.; the bronchus was severed through the extension (×10½).

TABLE II. SUMMARY OF DATA ON CASES IN WHICH EXTENSION HAD TAKEN PLACE

AGE	LUNG	SEX	TYPE OF TUMOR	GRADE	SITE OF EXTENSION	DISTANCE (MM.)
60	Right	Male	Squamous-cell epithelioma	3	Outer fibrous coat	1.3
51	Right	Male	Squamous-cell epithelioma	3	Outer fibrous coat	0.3
59	Right	Male	Squamous-cell epithelioma	3	Outer fibrous coat	1.4
59	Right	Male	Squamous-cell epithelioma	2	Outer fibrous coat	15*
53	Left	Male	Squamous-cell epithelioma	2	Submucosa	5*
54	Left	Male	Squamous-cell epithelioma	3	Outer fibrous coat	0.4
39	Left	Male	Squamous-cell epithelioma	2	Mucosa	5
55	Left	Male	Squamous-cell epithelioma	3	Outer fibrous coat	5
49	Right	Male	Squamous-cell epithelioma	3	Outer fibrous coat	8*
57	Right	Male	Squamous-cell epithelioma	3	Mucosa	0.8
57	Left	Male	Adenocarcinoma	3	Submucosa and outer fibrous coat	20*
60	Right	Female	Adenocarcinoma	3	Outer fibrous coat	1
57	Right	Male	Adenocarcinoma	3	Mucosa	8

*Carcinoma had extended to and apparently beyond the point at which bronchus was severed.

not surprising as adenocarcinoma is the most malignant of the types of carcinoma observed in these cases. In the case in which the adenocarcinoma had extended for 2 cm., the extension was divided when the involved bronchus was severed at operation. The extension had occurred in both the submucosa and in the outer fibrous coat. This is the only one of the thirteen cases in which proximal extension had occurred in more than one coat of the involved bronchus.

There was no evidence of proximal extension in any of the seven cases of adenoma. An adenoma of the bronchus grows slowly and rarely metastasizes. There likewise was no evidence of proximal extension in the case in which an indeterminate type of tumor was found.

It should be stated here that although involvement of the lymph nodes is as important in determining the prognosis as is extension of the malignant lesion in the bronchus, the lymph nodes were not studied. We felt an additional study of the lymph nodes would tend to confuse the picture and that this should be an entirely separate study.

No attempt was made to determine how long the patients survived post-operatively as this was felt to be irrelevant in a purely pathologic study.

It is important for the surgeon to know what type of tumor he is dealing with in order to know how close to the macroscopic lesion to make the operative incision. In this very limited number of cases it would appear that an adenocarcinoma extends much further on the average than does a squamous-cell epithelioma. With the skill of the modern bronchoscopist, biopsy can be done previous to the operation and the surgeon, knowing what kind of lesion he is dealing with, can decide where to sever the bronchus. Of course, there are certain anatomic limits and these probably accounted for the incision passing through the carcinoma in some of the cases in this series.

SUMMARY

This study is based on fifty-five cases of carcinoma of the lung in which the surgical specimens were examined microscopically to determine the proximal

extension of the malignant lesion along the bronchial wall. Only thirty of the fifty-five cases were included in the final survey. In the remaining twenty-five cases, the specimens had been mutilated by previous investigation or the carcinoma obviously did not involve a bronchus. The following types of tumor were encountered in the thirty cases: a squamous-cell epithelioma in sixteen, or 53 per cent; an adenocarcinoma in six, or 20 per cent; an adenoma in seven, or 23 per cent, and an anaplastic type of carcinoma in one, or 3 per cent, of the cases, respectively.

Although adenocarcinomas are considered more malignant than are squamous-cell epitheliomas, the tumor had extended proximally in ten, or 62 per cent, of the sixteen cases of squamous-cell epithelioma as compared with three, or 50 per cent, of the six cases of adenocarcinoma. The adenocarcinomas, however, extended proximally a greater average distance than did the squamous-cell epitheliomas. Of the sixteen squamous-cell epitheliomas, five were grade 2 and eleven were grade 3. Of the six adenocarcinomas, one was grade 2, four were grade 3, and one was grade 4. Proximal extension did not occur in the case in which the adenocarcinoma was grade 4. This is in agreement with the finding of New and Fletcher in cases of carcinoma of the larynx and with that of Conway and Broders in cases of carcinoma of the urinary bladder; namely, that carcinomas that are grade 4 are not as prone to spread by direct extension as are those that are grade 3.

There was no evidence of extension in the seven cases of adenoma or in the case in which an anaplastic type of carcinoma was encountered.

In eight cases extension occurred in the outer fibrous coat only; in three, it occurred in the mucosal coat only; in one, it occurred in the submucosa, and in one, it occurred in both the submucosa and the outer fibrous coat.

When dealing with adenocarcinomas the surgeon should, if anatomically possible, sever the bronchus more than 2 cm. proximal from the gross limits of the lesion. If the tumor is a squamous-cell epithelioma, a safe distance to sever the bronchus would be at least 1.5 cm.

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CLOSED EXTRAPLEURAL PNEUMONOLYSIS

A Preliminary Report

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THE extensive experience and reports of numerous surgeons have established closed intrapleural pneumonolysis as a safe and technically satisfactory procedure. Aside from hemorrhagic effusion and subcutaneous emphysema, the complications vary little from those occurring during the course of pneumothorax therapy in patients without adhesions. The procedure has a very low mortality.

There is less information, however, regarding the technical results and complications of extrapleural dissection of short pulmonary attachments and those in which portions of the lung are adherent over a relatively wide area. Coryllos and Ornstein,[3] Jones,[4] and Matson,[5] have described the extrapleural variation of intrapleural pneumonolysis but have not classified separately the results of those cases in which extrapleural dissection was used. There are still doubts in the minds of some operators as to whether this procedure is worth while. Turner[6] stresses the danger of the operation.

This report is based on fifty operations in which some degree of extrapleural dissection was performed. These represent all instances in which this procedure was used in 143 consecutive intrapleural pneumonolyses. No attempt has yet been made to evaluate the clinical results in this series because the pneumothorax treatment has not been completed.

A new type of sponge carrier and dissector* was used in forty-five of the fifty operations and has been described in a previous paper.[2] This instrument was developed because one of the early cases in this series had to be abandoned when the proper plane of dissection was obscured by a small amount of bloody oozing. The attachment had been partially separated by the cold cautery technique and no suitable instrument was at hand to wipe the area. This led to some experimentation with a method of applying woven cotton tape or gauze tape to the denuded area in much the same manner as a small pill sponge is used in the open operation for extrapleural pneumonolysis.

METHOD

Preoperative medication consisted of morphine sulfate, gr. ¼, or pantopon, gr. ⅓, with nembutal, gr. 1½, one-half hour before the procedure. The previous meal was omitted. After the shaving and application of alcohol while the patient was in the ward, he was painted with mercresin. A small exploratory cannula was then introduced. Through this a blunt Richardson pneumothorax needle was inserted in an attempt to palpate the lung. If the lung was found to be at a sufficient distance from the chest wall, an operating cannula was substituted and the location for the second cannula determined by thora-

From Glenn Dale Sanatorium, Health Department, District of Columbia.
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*May be obtained from American Cystoscope Makers, Inc., Lafayette Ave., Bronx, New York, N. Y.

coscopy. The cannulae were usually placed in the anterior and posterior axillary lines. In patients with bilateral pneumothorax or with considerable pulmonary fibrosis or disease on the contralateral side it was found advantageous to determine before operation whether the patient could withstand atmospheric pressure at his last pneumothorax refill. An extensive procedure is not justified if the patient is dyspneic.

The Coryllos pneumonolysis unit was used in nearly all cases. After all band adhesions were divided and the remaining attachment was closely inspected, the parietal pleura adjacent to the attachment was elevated with 1 per cent novocain solution and the pleura incised 1 to 2 cm. from the lung. From this point on, supplemented by further novocain infiltration, the attachment was separated from the chest wall by stripping with the new type of sponge carrier and dissector. Bleeding was controlled by the cautery. When dense fibrous strands appeared in the plane of cleavage the cautery was used for division after the strands had been clearly exposed. Small amounts of blood were wiped away by the dissector, larger accumulations being aspirated by means of a laryngeal suction tube and mechanical pump. All patients were fluoroscoped the next day and abnormal variations of the collapse corrected.

A peculiar situation arose on several occasions when postoperative fluoroscopy and x-ray seemed to indicate an immediate obliteration of the pneumothorax space at the site of dissection. No air space could be observed in that region even after rather large pneumothorax refills. After several weeks, however, the space reappeared and the pneumothorax proceeded normally. This artefact seems to have been due either to the presence of pools of blood which had clotted during the course of the dissection and remained in that region or to the appearance of fibrin over the denuded area. Since the presence of clotted blood is a factor in obliterative pleuritis we have sometimes used an aspirating tube of rather large bore in an attempt to withdraw clots.

OPERABILITY

In all extrapleural dissection two factors influencing the operability of attachments were kept in mind.

Type.—Short band adhesions which were unsafe to divide by the usual methods were found to separate extrapleurally without much difficulty. Inadequate retraction or unsatisfactory results from rotation of the patient, however, prevented division of the final layer of parietal pleura in some instances. When this occurred stripping of the pleura was carried toward the mediastinum for some distance. The resulting collapse was adequate although the postoperative x-ray appearance occasionally was peculiar (Fig. 5). Broad band adhesions extending toward the lung root were separated as far as was deemed safe.

Loose attachments, which appeared to be more common in the broader adhesions, gave little difficulty. They arose from the chest wall in a gentle curve. Those which arose with a sharp angle were usually dense and were separated with some difficulty. In this series an exploratory incision of the parietal pleura was made over an area of several centimeters. If the lung could not be separated with reasonable ease without frequent use of the cautery, the procedure was abandoned.

Location.—A cavity in the vicinity of the attachment was considered a definite contraindication because of the possibility that part of the blood supply to its wall might be parietal in origin and that loss of this supply might lead to necrosis and perforation of the lung surface.

No stripping was performed over the pericardium or diaphragm. Attachments in which one border was in the vicinity of the subclavian vessels were dissected toward the vessels and not completely divided (Figs. 1 and 2). Since the broader attachments are usually quite loose, the release of the greater part causes increased tension in the portion still attached. At reoperation the operator is surprised to find how often the stretched-out tissue can be transilluminated and divided even from the vessels themselves by the usual cauterization technique. Whenever the attachment involved a considerable portion of the subclavian vessels the procedure was not attempted (Fig. 3).

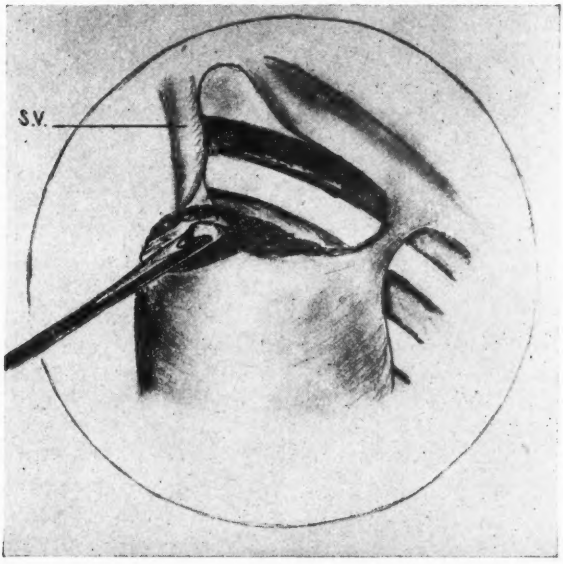

Fig. 1.—Stripping of an area of apical adhesion, one border of which is attached to the left subclavian vein.

The presence of tubercles on the lung was considered to be no contraindication to extensive stripping if they were several centimeters from the area of division. If they were on the parietal pleura, however, the operation was not attempted because it was felt that trauma might incite an empyema.

CLASSIFICATION OF DATA

In reporting intrapleural pneumonolyses it is a simple matter to classify the operations as those in which all adhesions were divided and those in which only some of the adhesions were completely divided. When considering extrapleural pneumonolyses by the closed method, however, the operations must be classified somewhat differently. Here they are grouped according to whether the separation of the adhesion was *partial, extensive,* or *complete.* Those in which *partial* dissection

was done were exploratory in character and the operation was terminated because of the density of the attachment. There were thirteen of these procedures in the group to be reported. In one case the procedure was performed on three occasions before a satisfactory collapse was obtained.

Those operations considered *extensive* in character accomplished a broad separation without complete division. Some lungs were left with a strip of free parietal pleura attached because of the technical difficulty

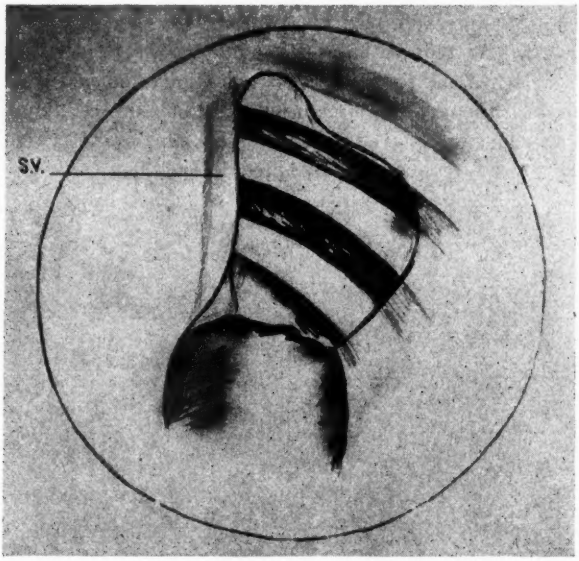

Fig. 2.—Dissection completed. A strip of parietal pleura is still attached to the subclavian vein. This strip may be divided at a second operation. In this instance it was not necessary.

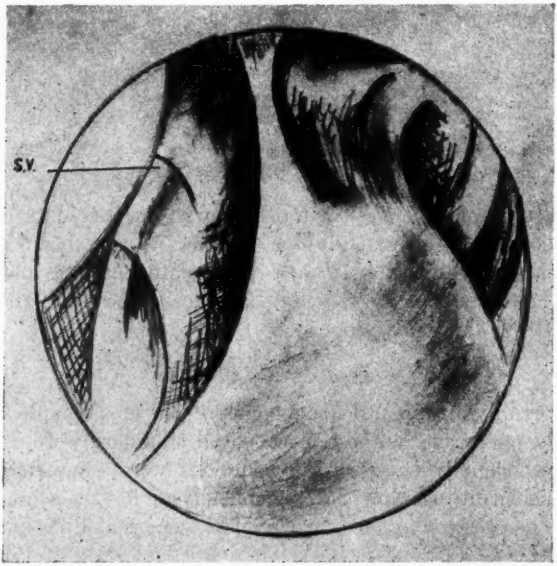

Fig. 3.—This dense attachment overlying a portion of the subclavian vein is inoperable by this method.

of further division. The other dissections were carried as close to the mediastinum or vessels as was considered safe. There were twenty-two in this group.

In the *complete* division group it was possible to enucleate the attachment completely. There were fifteen in this group.

In all, fifty extrapleural procedures were performed on forty-six patients. Sixteen of the attachments were short bands, the remainder being surface-to-surface attachments up to four interspaces in width.

With regard to the technical results, only those operations were considered satisfactory which succeeded in freeing an area of lung sufficient to permit an adequate collapse. This was checked by x-ray.

TABLE I

RESULTS AND COMPLICATIONS OF FIFTY CLOSED EXTRAPLEURAL PNEUMONOLYSES

	PARTIAL DISSECTION		EXTENSIVE DISSECTION		COMPLETE DISSECTION		
	BROAD ADHERENCE	SHORT BAND	BROAD ADHERENCE	SHORT BAND	BROAD ADHERENCE	SHORT BAND	TOTAL
Operations attempted	13	0	18	4	3	12	50
Satisfactory collapse	4		14	4	3	10	35
No postoperative fluid	7		11	1	1	6	
Slight postoperative fluid	3		7		1	4	
Effusion					1		
Bloody effusion	3			2		1	
Late tuberculous empyema			2	1	1	1	
Temperature over 100.4° F.	5		8	3		4	
Staphylococcus aureus empyema				1			
Bronchopleural fistula 9 months later			1				
Spontaneous pneumothorax						1	

RESULTS

Exploratory *partial* dissection was performed thirteen times. One patient had three partial operations. Four of the thirteen operations (four of eleven cases) produced an anatomically satisfactory collapse. All of the attachments in this group were surface-to-surface in type. Five operations were followed by elevations of temperature over 100.4° F. Three patients developed a small amount of clear fluid. Postoperative hemorrhage of 1,600 c.c. in one case was removed by aspiration with transfusion vacuum bottles and the filtered citrated blood reintroduced into the median basilic vein. Bloody fluid up to 1,500 c.c. accumulated over a period of several days in two other instances.

Extensive dissection was performed twenty-two times on twenty-one patients. In fourteen of the eighteen operations upon surface-to-surface attachments the resulting collapse was anatomically satisfactory. Four broad band adhesions were extensively separated and in each instance a satisfactory anatomic collapse was achieved. Eleven of the total of twenty-two operations in this group were followed by elevations of temperature over 100.4° F. Seven had a small amount of fluid. Two instances of bloody fluid, 500 and 700 c.c., respectively, followed the procedure. One patient developed *Staphylococcus aureus* empyema. Tuberculous empyema developed four months later in one instance, six months later in another case, and seven months later in a third, the

last followed by a bronchopleural fistula nine months after operation. The patient developing an empyema seven months after operation was the only one in this series exhibiting subpleural tubercles on the lung. These were at some distance from the point of dissection.

Complete division of the attachments was performed fifteen times. Complete division of surface-to-surface attachments was possible in only three instances and a satisfactory collapse followed in each case. Twelve band adhesions were completely enucleated producing ten satisfactory collapses, the other two failing because of other inoperable adhesions. In this group of fifteen cases, four patients had temperature elevations, four had small postoperative effusions, and one developed a bloody effusion of 950 c.c. A tuberculous empyema occurred five months later in one case.

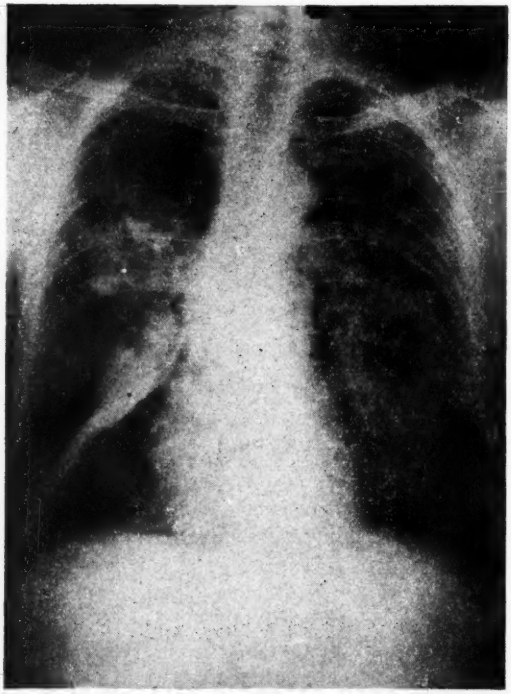

Fig. 4.—Case 1.

ILLUSTRATIVE CASE REPORTS

CASE 1.—A 48-year-old white man was admitted July 26, 1943, with far-advanced tuberculosis. A film of the chest showed the upper three-fourths of the right lung filled with a dense, nearly completely opaque caseofibrous infiltration. There were several cavities in this region. The left side showed a light nodular infiltration in the lung field between the first and fourth anterior ribs.* The sputum was Gaffky 2 on direct smear. An x-ray taken Oct. 6, 1943, showed no improvement.

Right pneumothorax was instituted Oct. 18, 1943. Bronchoscopy Nov. 19, 1943, showed mucosal injection of the lower portion of the right

*From the report of Dr. Walter H. Merrill, July 30, 1943.

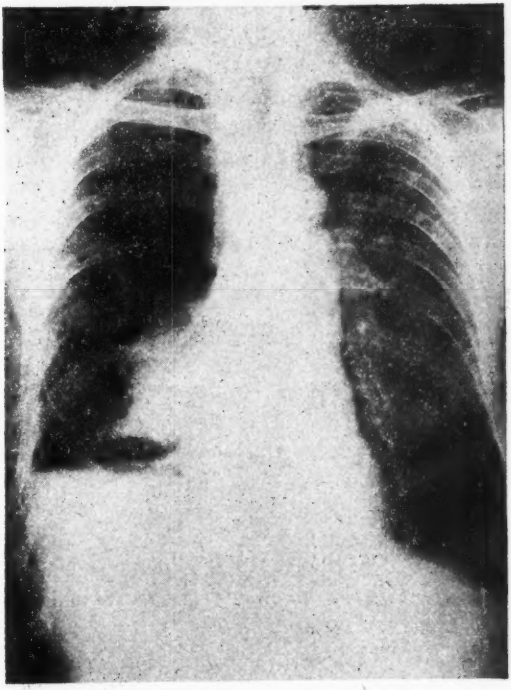

Fig. 5.—Case 1.

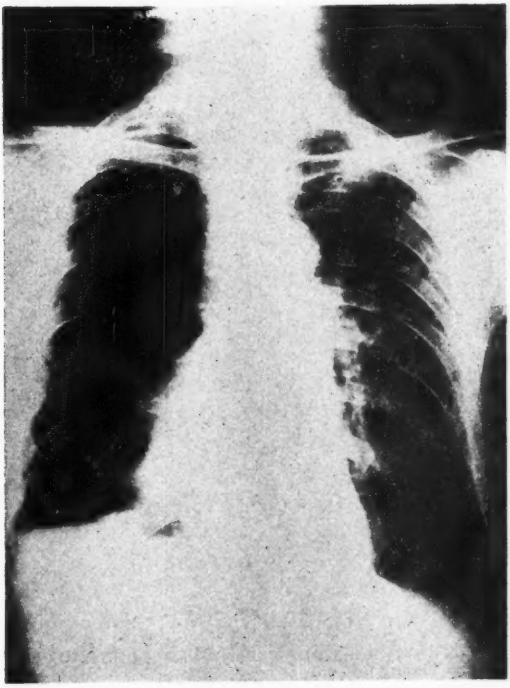

Fig. 6.—Case 1.

main stem bronchus with general narrowing of the lumen. Twenty-five per cent silver nitrate was applied. An x-ray Nov. 12, 1943, showed an extensive pleural adhesion (Fig. 4).

Nov. 24, 1943, a right pneumonolysis was performed at which time one narrow band was divided and a broad attachment in the posterior axillary line, roughly 7 by 15 cm. in size, separated to the lung root by combined cauterization and extrapleural dissection. Because the attachment was not completely enucleated the operation was classified as *extensive*.

Sputum became negative Dec. 13, 1943. A small amount of clear fluid had been present on several occasions since operation, disappearing spontaneously. On the x-ray film of March 9, 1944 (Fig. 5), a peculiar annular shadow the size of a five-cent piece was present. This had almost disappeared on the x-ray of May 5, 1944 (Fig. 6). The shadow was considered to be due to the configuration of edges of the free parietal pleura as it retracted upon the collapsed lung. No subpleural blebs had been noted at operation.

Shortly after the film made in May, a single positive sputum appeared and the patient again was found to have tracheobronchitis. Tuberculous laryngitis appeared but has been subsiding. At present the sputum is negative and there are no symptoms of tracheobronchitis.

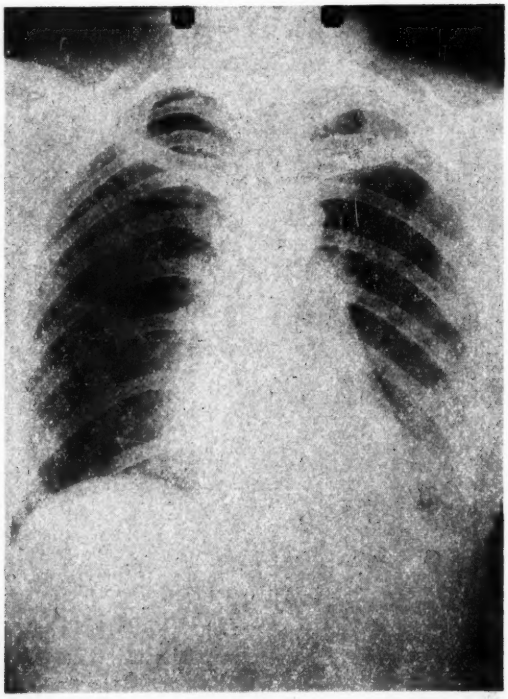

Fig. 7.—Case 2.

CASE 2.—A 33-year-old white woman was admitted Feb. 21, 1941, with an exudative lesion of the upper one-fourth of the right lung and the apex of the left. Sputum was Gaffky 7 on Oct. 27, 1941, and a right

phrenic crush was performed. June 20, 1942, the sputum was again positive. A right pneumothorax was started Oct. 13, 1942, and produced only a fair collapse because of extensive adherence of the lung to the cupola (Fig. 7). Dec. 29, 1942, a *partial* extrapleural separation of an extensive surface-to-surface attachment was performed, producing only slight improvement in the collapse. Four days later, 1,200 c.c. of bloody fluid were removed. A second *partial* dissection was performed Jan. 26, 1943, and a third operation Feb. 23, 1943, at which time nearly complete separation of the entire apex was accomplished. During the last operation it was found necessary to divide a portion of the periosteum of the third rib and the third intercostal nerve before a satisfactory separation could be achieved. Three hundred cubic centimeters of bloody fluid were aspirated following the last procedure and none has appeared since then. The collapse at present is satisfactory (Fig. 8) and sputum remains negative.

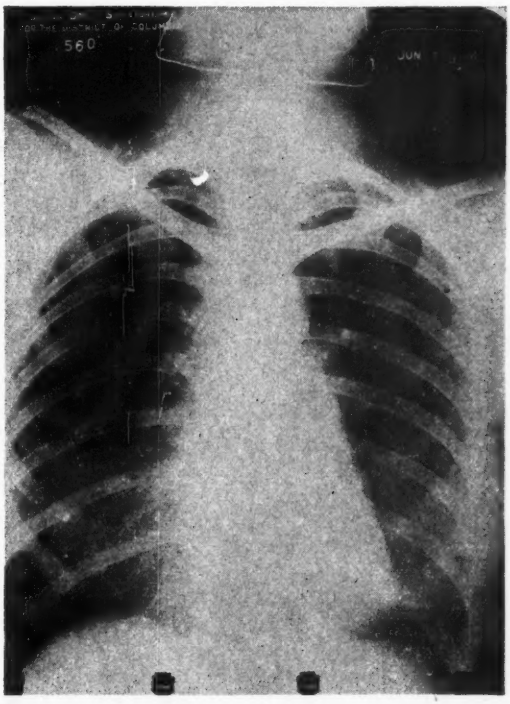

Fig. 8.—Case 2.

Without operation this patient would have been under collapse therapy for an indefinite period with a very poor prognosis in spite of the negative sputum. Her attachments were somewhat difficult to separate. She had a considerable amount of bloody fluid following the first attempt, but the resulting improvement in the collapse suggested further investigation. An area of dense fibrosis involving the periosteum and an intercostal nerve appeared to be the main origin of the attachment. Since relatively small areas were enucleated at each operation they were considered *partial* in extent.

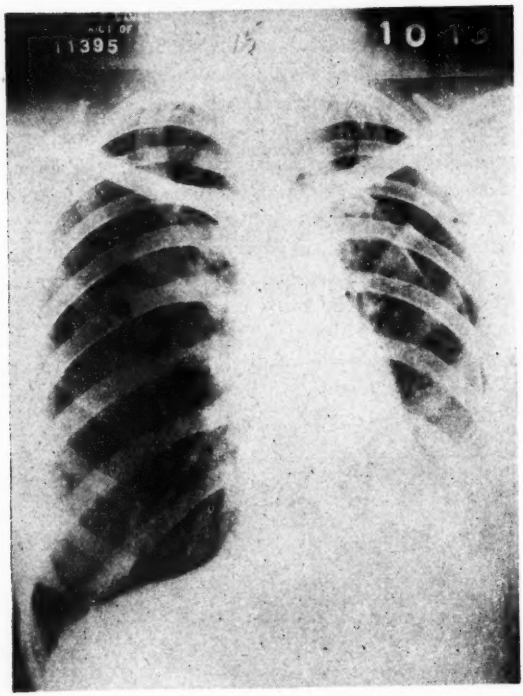

Fig. 9.—Case 3.

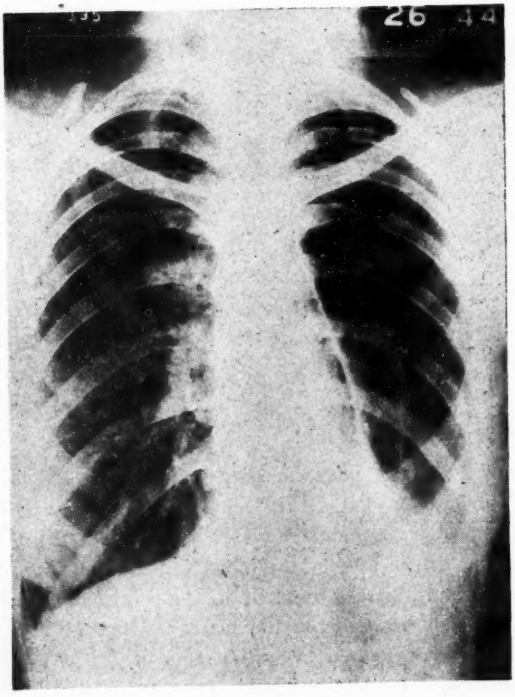

Fig. 10.—Case 3.

CASE 3.—A 22-year-old white woman was admitted Oct. 22, 1941, with a moderately advanced lesion involving the upper one-fourth of the right lung field and upper one-third of the left lung. Sputum was Gaffky 3. Left pneumothorax was induced Nov. 6, 1941. Left pneumonolyses were performed Jan. 5 and April 28, 1942, and satisfactory collapse obtained. In March, 1943, x-ray examination disclosed an increase in the amount of tuberculosis at the right apex although sputum remained negative for acid-fast bacilli. Sputum was blood streaked on April 5, 1943. A gastric specimen, July 2, 1943, was positive and the left pneumothorax was allowed to re-expand partially. A small right spontaneous pneumothorax occurred July 28, 1943 and was maintained. Definite cavity formation at the apex of the right lung was then clearly apparent (Fig. 9). Oxygen lavage was performed on the left side to hasten re-expansion. Considerable difficulty was encountered in increasing the right pneumothorax pressure to neutral without producing dyspnea. This was achieved Oct. 29, 1943, and it was possible to perform a right pneumonolysis without discomfort.

The apex of the lung was found to be suspended by a complex band adhesion extending from a surface-to-surface attachment over the posterior portion of the cupola. By anesthetizing the base of these attachments with novocain solution and by combined cauterization and extrapleural dissection the entire apex was freed.

It was necessary to withdraw air from the right pleural cavity the next day until pressure readings were −14 and −4. Since then the patient has withstood the bilateral pneumothorax without difficulty (Fig. 10). A small amount of fluid disappeared postoperatively and it has been possible to increase the right pneumothorax. Sputum and gastric examinations have been negative since the right pneumonolysis was performed.

This case presented an unfortunate situation—an unstable left lung lesion temporarily controlled by a pneumothorax, a poor right pneumothorax in which a cavity was still apparent, and a history of right spontaneous pneumothorax. Fortunately, the cavity was some distance from the pleural attachment and the entire apex could be completely freed. In cases of this type, as Coryllos and Ornstein[3] mention, heroic measures only can be considered as offering a reasonable chance for success. The procedure was carried out with that attitude in mind.

CASE 4.—A 42-year-old Negro woman was admitted Aug. 17, 1942, with an extensive caseopneumonic lesion on the left side and fibrocaseous infiltration at the right base. The sputum was positive for acid-fast bacilli. Left pneumothorax was induced Aug. 28, 1942, and found to be complicated by apical adhesions (Fig. 11). On Oct. 23, 1942, a left pneumonolysis was performed. A short, dense band adhesion was found at the level of the second rib in the posterior axillary line. This was divided by extrapleural dissection. The upper lobe was then found to be adherent on the costovertebral gutter from the apex to the seventh rib. Investigation of this attachment revealed that it was made up of numerous veil-like bands which could be separated by merely drawing the lung forward with the cloth-covered dissector. It was possible to strip the lung to the spine before local irritation and coughing from the manipulation became very severe (Fig. 12). Very little oozing

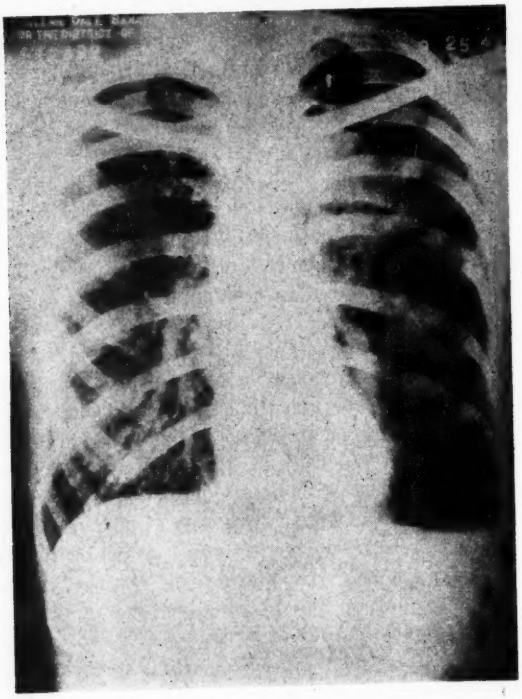

Fig. 11.—Case 4.

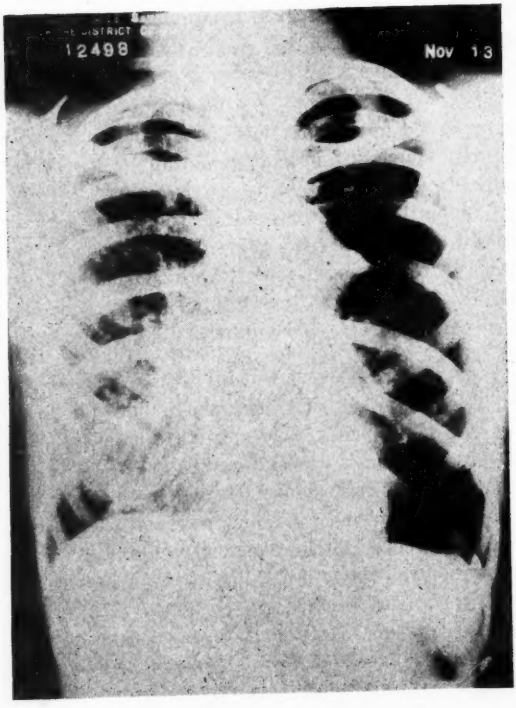

Fig. 12.—Case 4.

occurred. No fluid followed. Sputum was blood streaked ten days after operation but was controlled by increasing the extent of the pneumothorax. Pneumothorax has been continued to the present. The last positive sputum specimen was on Nov. 16, 1942, and serial x-rays indicate progressive resolution of the lesion at the right base.

This case indicates the value of having an instrument at hand which can separate thin veil-like attachments by merely stretching them. A cautery tip or other metal applicator may perforate the parietal pleura during manipulation. Here the traction afforded by the cloth applicator was both a safeguard and an advantage.

SUMMARY AND CONCLUSIONS

The fifty cases of extrapleural dissection presented represent 35 per cent of 143 intrapleural pneumonolyses. The attachments in these fifty cases were unsuitable for division by the accepted criteria for intrapleural pneumonolysis and under ordinary circumstances would not have been explored. It was possible to obtain satisfactory collapse in 35 or 70 per cent of them by extrapleural dissection. An extensive or complete separation was achieved in thirty-seven cases. If we omit the number performed for exploratory purposes (thirteen) the operation succeeded in thirty-one of thirty-seven cases, or in 84 per cent of the cases, in which an *extensive* or *complete* dissection was possible.

No postoperative fluid was noted in twenty-six cases. Transient effusion occurred in fifteen instances. Twenty operations were followed by temperatures over 100.4° F. which lasted no more than one week except in the instances in which bloody effusion and the staphylococcus empyema occurred. The latter situation was probably the result of damage to lung parenchyma in the extremely firm short band. Closed drainage was necessary.

Considerable postoperative bleeding followed the operation in three of the patients who had *partial* dissection (23 per cent) and in three in the other two groups (8 per cent). These percentages suggest the various nature of the attachments. Those attachments which after exploration are found to be too dangerous to dissect also appear to be the ones which are associated with the greater percentage of postoperative hemorrhagic effusion even though the operation is not persisted in. When extensive or complete dissection is possible, postoperative oozing is rather infrequent. Dense attachments are frequently vascular and it is suggested that the normal respiratory tug at the denuded line of attachment exaggerated by coughing and occasional vomiting has the opportunity for rupturing vessels and loosening the coagulum following cauterization of bleeding points.

There were six cases of empyema, five being tuberculous, all of which developed four or more months after the procedure. One *Staphylococcus aureus* empyema occurred postoperatively. Alexander[1] reports various percentages of tuberculous empyema in the pneumothorax cases of a number of authors and mentions Hayes with 21 per cent, L. S. Peters with 20 per cent, and Kinsella with 10 to 12 per cent. The incidence of tuberculous empyema here reported (10 per cent) is, therefore, not excessive.

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ABNORMAL ESOPHAGEAL COMMUNICATIONS: THEIR TYPES, DIAGNOSIS, AND THERAPY

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AN ESOPHAGEAL fistula is admittedly a precarious clinical condition but is not necessarily incompatible with life. Interest in this condition was aroused by a recent group of cases which portrayed different types of abnormal esophageal communications. There has been a considerable amount of literature on certain types of these abnormalities and much interest has been aroused by the successful handling of congenital tracheo-esophageal fistulas by the work of Haight and Towsley[1] and Ladd.[2] Prior to their work such anomalies spelled an inevitable doom. The acquired types of fistulous communications have been described fairly frequently since the original communication of McDonnell[3] in 1844. However, the reports were concerned mainly with autopsy findings and it was not until the advent of x-ray contrast media, the bronchoscope, and the recent advances in thoracic surgery that interest in this condition could be aroused from the angle of a therapeutic challenge. It is of utmost importance that at last equal attention must be paid to the nutritional support of the patient as to the diagnostic and specific therapeutic procedures. The elimination of the use of barium when such conditions are suspected would in itself be a significant aid to the patient's prognosis. Iodized oil is the contrast media of choice used.

These lesions have always been classified into two main groups, namely, the congenital and the acquired. There has been such adequate description of the congenital abnormalities in the recent literature that it is not felt necessary to discuss these further in this communication. The acquired types must be subdivided according to the area with which the abnormal communication of the esophagus exists. The most frequently encountered one is a communication between the tracheobronchial tree and the esophagus. Next in frequency would be openings onto the skin surface. Such tracts may go devious routes such as are seen in the neck, in the chest by way of the pleural cavity and occasionally through the mediastinum and present at a considerable distance away on the body surface.[4] There have been two cases of communication between the esophagus and the pericardial cavity at the Barnes Hospital but descriptions of them have not been encountered in the literature.

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In summary, then, one may have an esophagotracheal or esophagobronchial fistula, an esophagocutaneous, esophagopleural, esophagopleurocutaneous, and esophagopericardial fistula.

The etiology of such conditions depends upon the same factors which may cause fistulas elsewhere, namely, malignant tumors, inflammatory diseases, trauma, and possibly predisposing congenital abnormalities. Carcinoma of the esophagus, bronchial tree, and malignancies of neighboring structures such as the thyroid[5] and mediastinal glands constitute the most frequent cause for esophageal fistula formation. Thus, Monserrat[6] found 367 fistulas of neoplastic, 222 of congenital, 41 of infectious, and 40 of traumatic origin. The most common infectious etiology is undoubtedly tuberculosis. Another chronic inflammatory cause is syphilis. Ono and Bucher[7] (1934) described twelve cases due to syphilis, Moersch and Tinney[5] (1944) added four more of syphilitic origin, and further cases of the same origin are included in the case reports to be presented. One case due to actinomycosis was described by Vinson and Sutherland[8] in 1926. It is remarkable that this patient lived for twenty years after the development of the fistulous tract. A fistula may develop in conjunction with an esophageal diverticulum. Such a diverticulum is most likely to be of the traction or acquired type, so that the diverticulum itself plays a secondary role in the actual etiology. In a report[5] from the Mayo Clinic of thirty-eight cases, 13 per cent were due to trauma. The types of trauma which are found reported included gunshot wounds, the ingestion of sharp objects, crushing injury to the chest, penetration by bone fragments, and surgical procedures, such as bougie dilatation, esophagoscopy, and anastomoses.

In discussing the mode of origin of lesions of an inflammatory nature, it is often difficult to determine whether the esophagus ruptured from within or was eroded from without. Cases have been described in which a tuberculous ulcer was present in the esophagus and considered as the site of origin.[9, 10] Other cases are described in which it was felt that a diseased lymph node with the formation of a traction diverticulum and final erosion into this signified extrinsic origin.[11] Sindell[12] described fourteen instances among 114 cases of empyema necessitatis in which the empyema cavity drained into the esophageal lumen. Spontaneous rupture of the esophagus has been noted in association with alcoholism (Williams and Boyd),[13] vomiting (Lewis and Smeed),[14] purgation and severe sepsis (Gott).[15] Vascular thrombosis, infarction, cardiospasm, ulceration, esophagitis, stricture formation, have all been described in conjunction with spontaneous perforation.

The symptomatology and physical findings are of necessity quite varied according to the site and type of fistula as well as the etiology. Prominent symptoms which have been noted include dysphagia, vomiting of purulent material or of an empyema irrigant material, the transformation of a hydrothorax into a hydropneumothorax, and the appearance of food particles in the sputum. By far the most suggestive complaint is that of coughing paroxysms and dyspnea following the ingestion of food. There is usually a marked cachexia because of the distress coincident with attempts at deglutition.

The ease with which a diagnosis of this condition is made depends in large part upon the site. In those in which a cutaneous element exists, the appearance of food particles or ingested dyes such as methylene blue or gentian violet is of obvious significance. The finding of numerous yeast cells in empyema fluid may be of diagnostic import.[16] In such cases, dyes may be used to enhance the

diagnosis. The finding of tracts following the ingestion of iodized oil is probably the most valuable diagnostic sign. The esophageal stoma may be seen at esophagoscopy, but this may be difficult to define. In cases of communications between the esophagus and tracheobronchial tree, one may find the bronchial opening with the bronchoscope after placing dye into the esophageal lumen. An unusual case was reported by Judd and co-workers[17] in which a swallowed string went through the fistula and wrapped itself around the hypopharynx. The transformation of a hydrothorax into a hydropneumothorax, although most commonly due to a bronchopleural fistula, can be caused by an esophagopleural fistula. The appearance of air and fluid in the pericardial sac, in a patient with known esophageal disease, may signify the presence of a fistula.

In regard to therapy, the fundamental problems are the maintenance of nutrition and adequate drainage. It is quite obvious that a slowly formed fistulous tract may be compatible with life if adequate nutrition is maintained. Indeed, in one case[18] the patient's ability to feed himself with the aid of a Levin tube for thirty-two months prior to local therapy serves to emphasize this premise. Other cases with a survival up to seven years with the addition of either spontaneous or induced drainage have been reported. Spontaneous cure by drainage alone is not to be expected except in conjunction with some form of physiologic rest for the esophagus. That is to say, if one uses either a gastrostomy or an indwelling Levin tube for feeding. Such cures are encountered in the cases wherein the communication is with an empyema cavity. Such empyema needs to be of the nontubercular variety except in the rare instance. Although no such cases have been reported, one would suspect that the use of an Abbott-Rawson tube would be of considerable value in maintaining a dry esophagus and an adequately nutritioned stomach. In the cases wherein the communication lies between the esophagus and tracheobronchial tree, and such communication is not due to cancer, some form of direct attack appears to be indicated. Several reports of the use of silver nitrate applications, even in high concentration, have been unsuccessful. The marked stenosing effect of lye has led to the successful application of pure sodium hydroxide in three reported instances.[17, 18] In the cases of esophagopleural fistulas, a successful result demands obliteration of dead space, either by adequate lung re-expansion or by thoracoplasty. Cases of direct surgical attack have been reported with success.[17-19] Such attack varies with the location and in the high esophagotracheal fistulas, transection of the tract and interposition of tissue with or without closure of the stomata have been curative. Local resection of lung tissue and adequate drainage with or without closure of the esophageal opening has been known to heal the disease. No reports exist in regard to successful esophagopericardial communication. Upon reading the literature we find that a total of fifteen cases of esophageal fistulas, for which a permanent closure was obtained by any method, have been collected.[16-25]

REPORT OF CASES

In reviewing the cases of abnormal esophageal communications which have been handled at the Barnes Hospital, one is struck by the conformity of these cases with the groups reported in the literature. The great predominating cause is found to be malignant lesions of the esophagus or larynx, constituting thirteen of the twenty-two cases herein reported, while a fourteenth instance occurred in association with Hodgkin's disease. Fistulas secondary to chronic empyemas constitute two cases, two were secondary to operative procedures, while an additional two were in relation to syphilitic aortic aneurysms. One patient developed a

fistula following instrumentation, while another developed one in association with a congenital diverticulum of the esophagus and instrumentation had occurred in this patient. No examples of fistulas in relation to tuberculous empyema or tuberculous mediastinal lymphadenitis are contained in this report. This is due at least in part to the fact that patients with chronic tuberculosis are usually relegated to sanatoriums.

The cases to be reported are listed in Table I. To clarify further and emphasize certain features, individual cases are reported in detail to exemplify one fistula secondary to each cause. The two examples of esophagopericardial communications are given in detail because of their apparent extreme rarity. The patient whose esophageal defect occurred in association with Hodgkin's disease is individually detailed for the same reason.

MALIGNANT LESIONS

CASE 1 (B. H. No. 13277).—D. J., a 46-year-old negro man, came to the hospital in July, 1922. His chief complaints were a productive cough for five months, sore throat for four months, dyspnea for two months, voice loss for one month, and weight loss of fifteen pounds. For three weeks, choking paroxysms had been noted on taking liquids. Physical findings revealed emaciation, orthopnea, excessive salivation, and a fixed hard tumor mass involving the right lobe of the thyroid. A gastrostomy was done, July 17, and local x-ray therapy given but the patient expired July 25. It is interesting to note that the patient had exacerbations of dyspnea with each gastrostomy feeding. Post-mortem examination recorded a primary esophageal carcinoma with metastases in the thyroid, right lung, clavicle, pleura, peribronchial, mediastinal, and mesenteric lymph nodes. An ulcerated area 3 by 1 cm. was found on the anterior esophageal wall 18 cm. below the glottis with peri-esophageal tumor mass in this region and a tracheobronchial fistula which admitted a probe easily.

CASE 2 (B. H. No. 30328).—M. M. K., a 36-year-old white woman, was admitted, August, 1933, complaining of nonproductive cough, recurrent choking spells, and hoarseness. Physical examination revealed diffusely nodular, enlarged thyroid. A round shadow in the right lower paramediastinal area was noted on fluoroscopy. The only positive finding noted on bronchoscopic examination was a paralysis of the left vocal cord. A course of x-ray therapy was instituted. Two months later the patient was readmitted and a large cervical abscess extending down into the anterior mediastinum was drained of 150 c.c. thick pus. Marked improvement occurred but a persistently draining sinus in the suprasternal notch area was found present one month later. This tract was explored, biopsied, and more adequate drainage instituted. The biopsy revealed nonspecific chronic inflammatory reaction. In December, 1934, she returned complaining of markedly productive cough, fever and coughing paroxysms, and continued drainage from the cervical sinus tract. Bronchoscopic examination showed evidence of extrinsic tracheal compression and excessive mucopurulent secretions throughout the bronchial tree. An exploration of the anterior mediastinum was performed (by E. A. G.) and revealed a diffuse mass of chronic inflammatory tissue. Postoperatively she became more troubled by severe coughing paroxysms and these were found to be associated with drinking fluids. Barium x-ray studies, Jan. 21, 1935, demonstrated a tracheoesophageal fistula at the level of the suprasternal notch. She would not tolerate an indwelling nasal tube so a gastrostomy was performed Jan. 24, 1935, and a simultaneous cervical lymph node biopsy demonstrated the presence of Hodgkin's disease. Radiation therapy was given. March 9, 1935, combined bronchoscopic and esophagoscopic examination was performed, and methylene blue applied to the anterior esophageal wall was seen to project through one ulceration into the trachea 4.5 cm. above the carina. This area was treated with 50 per cent acriflavine solution. Progressive improvement occurred and one month later (April 12, 1935) barium studies failed to reveal the presence of a fistula. The gastrostomy was allowed to close and oral feedings were well tolerated. She did well for six months after which the Hodgkin's disease began to flare up and despite recurrent x-ray therapy, the course was progressively downhill, resulting in death, July 18, 1936. Post-mortem examination revealed diffuse Hodgkin's infiltration of thoracic and abdominal lymph nodes, both lungs, the spleen, and adrenal glands. Marked fibrosis of the upper mediastinum was noted, while the only remaining evidence of the pre-existing tracheo-esophageal fistula consisted of healed ulcers on the anterior esophagus and posterior trachea at the level of the suprasternal notch. Microscopic sections taken through this area revealed old hyalinized scar tissue only.

TABLE I

PATIENT	AGE	SEX	TYPE OF FISTULA	ETIOLOGY	TREATMENT	RESULT
1. O. M. C. (U1897)	4 yr., 8 mo.	F	Esophagopleural	Chronic empyema	Thoracotomy drainage, nasal tube feedings	Cured
2. W. D. G. (30285)	32 yr.	M	Esophagopleural	Lung abscess, chronic empyema	Thoracotomy drainage, nasal tube feedings	Died of brain abscess
3. C. J. C. (52602)	33 days	F	Esophagotracheal, esophagopericardial	Congenital diverticulum	Nasal tube feedings	Died
4. H. H. K. (111650)	46 yr.	M	Esophagopericardial	Pulsion diverticulum, postoperative empyema	Pericardiotomy, jejunostomy	Died of pulmonary embolism
5. R. L. E. T. (12524)	54 yr.	M	Esophagomediastinal	Syphilitic aortic aneurysm, mediastinal abscess		Died
6. W. H. (69655)	64 yr.	M	Esophagotracheal	Syphilitic aortic aneurysm, secondary traction diverticulum	Levin tube feedings	Died
7. T. M. (118953)	74 yr.	M	Esophagopleural	Surgical excision of carcinoma, esophagogastric juncture	Thoracotomy drainage, jejunostomy	Cured
8. J. G. H. (94713)	47 yr.	M	Esophagobronchial	Instrumentation	Lobectomy, thoracotomy drainage, nasal tube feedings	Died
9. M. M. K. (30328)	36 yr.	F	Esophagotracheal	Hodgkin's disease	Chemical cauterization, x-ray therapy	Cured; died 16 mo. later
10. D. J. (13277)	46 yr.	M	Esophagotracheal	Carcinoma of esophagus	Gastrostomy and x-ray therapy	Died
11. T. E. S. (81640)	59 yr.	M	Esophagotracheal	Carcinoma of esophagus	Gastrostomy	Died
12. C. E. S. (38702)	63 yr.	M	Esophagotracheal	Carcinoma of esophagus	Moribund on admission	Died
13. D. DeV.	65 yr.	M	Esophagotracheal	Carcinoma of esophagus	Moribund on admission	Died
14. J. G. K. (15816)	53 yr.	M	Esophagotracheal	Carcinoma pyriform fossa	Gastrostomy	Died
15. E. W. (25687)	37 yr.	M	Esophagotracheal cutaneous	Carcinoma of larynx	Laryngectomy, radium therapy	Died
16. S. G. McK. (36999)	52 yr.	M	Esophagotracheal cutaneous	Carcinoma of larynx	Laryngectomy	Fistula cured
17. D. M. (5711)	12 yr.	M	Esophagotracheal	Carcinoma of larynx, tracheotomy	Surgical repair, tracheotomy and gastrostomy and surgical repair	Died three years later
18. M. J. K. (23209)	52 yr.	M	Esophagotracheal	Carcinoma of esophagus	Gastrostomy	Died
19. D. G. B. (1047)	61 yr.	M	Esophagobronchial	Carcinoma of esophagus	Gastrostomy	Died
20. J. R. K. (47726)	45 yr.	M	Esophagotracheal	Carcinoma of esophagus	Gastrostomy	Died
21. H. R. (32569)	57 yr.	M	Esophagocutaneous	Carcinoma of larynx, cervical abscess	Laryngectomy, surgical repair	Cured
22. J. R. (39173)	45 yr.	M	Esophagotracheal	Carcinoma of esophagus	Gastrostomy	Died
23. G. K. A. (69098)	51 yr.	M	Esophagopleural cutaneous	Postpneumonectomy, empyema, carcinoma of lung	Abstinence from fluid intake, improved thoracotomy drainage	Cured

ESOPHAGOPLEURAL FISTULA (SECONDARY TO EMPYEMA, NONTUBERCULOUS)

CASE 3 (St. L. C. H. No. U1897).—O. M. C., a 4-year-old white girl, was first seen at the St. Louis Children's Hospital Oct. 18, 1944. There was a history of intermittent cough for the past two years following a pneumonia complicated by an empyema. This empyema had been treated elsewhere by needle aspiration only. A marked flattening deformity of the

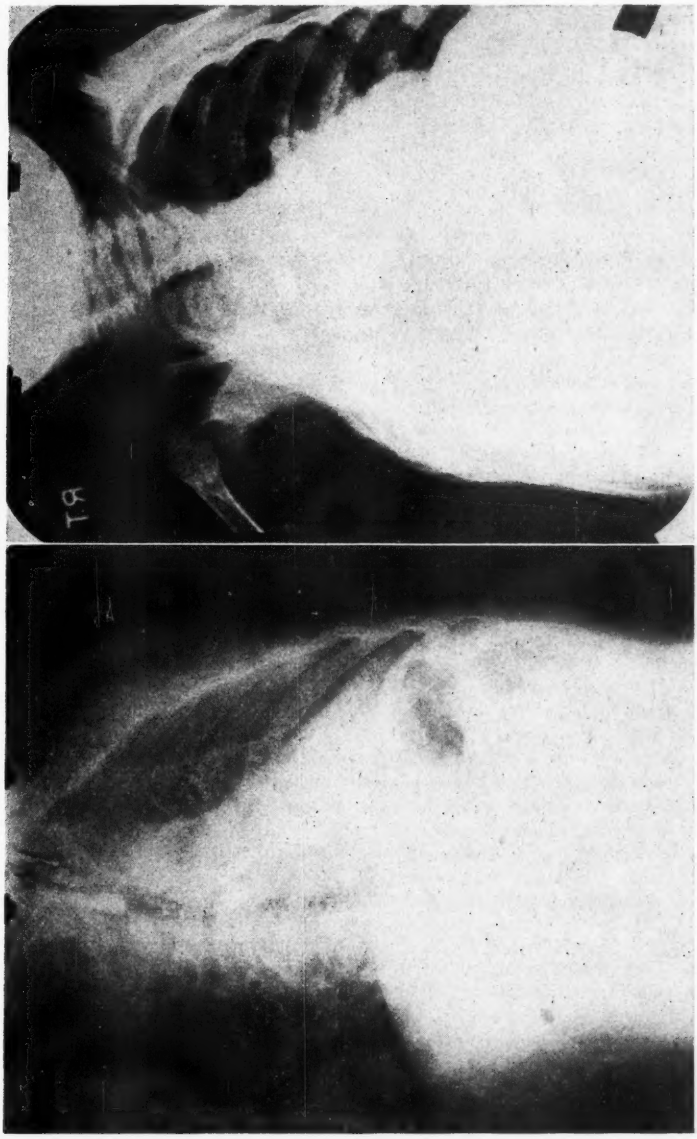

Fig. 1.

Fig. 2.

Fig. 1 (Case 1).—D. J. Lipiodol x-ray study showing the communication between the esophagus and left bronchus. Some evidence of the associated bronchiectasis is demonstrated.

Fig. 2 (Case 3).—O. M. C. X-ray taken on admission showing deformity of right chest cage secondary to chronic empyema, which was present for two years prior to discovery of two esophagopleural fistulas.

right anterior part of the chest had been present for one year accompanied by a failure to grow or gain weight. An episode of vomiting and diarrhea finally brought the patient to the care of the referring physician, who did a barium x-ray study and which revealed two pleuro-esophageal fistulas. Examination revealed an acutely and chronically ill child, extremely underweight (twenty-one pounds at 4 years and 8 months of age). The chest deformity so often seen in neglected empyemas in children was noted on the right side. Breath sounds were absent

over the right side of the chest. X-ray of the chest revealed an homogeneous opacity throughout the right lung field. Open thoracotomy tube drainage was instituted October 21. Methylene blue ingested by mouth was found to appear immediately through the thoracotomy tube. The patient's nutrition was maintained by utilizing Levin tube feedings for three weeks. The right lung was noted to partially re-expand, and on November 12 it was no longer possible to demonstrate the presence of a fistula by oral ingestion of dye. Oral feedings were therefore started. The patient progressively and rapidly improved. She gained ten pounds in four months. The thoracotomy tube was permanently removed, March 15, 1945. She has been followed for three months thereafter and has continued without evidence of recurrence. Bacteriologic studies on the empyema fluid revealed *Streptococcus viridans*. Roentgenologic studies done in February revealed no further evidence of fistula but diverticula were present. It should be noted that repeated tuberculin tests were consistently negative.

ESOPHAGOMEDIASTINAL FISTULA IN RELATION TO SYPHILIS

CASE 4 (B. H. No. 12524).—R. L. E. T., 54-year-old white man, was first seen Sept. 13, 1927. He gave a history of progressive dysphagia for three months, culminating in total esophageal obstruction ten days prior to entry. He had had an hematemesis in 1922, and an aortic aneurysm had been noted at that time; he received some antisyphilitic therapy. Substernal pain had been noted for three and one-half years. Severe chest pain associated with extreme dysphagia had been noted, August 29, and had increased for six days when a severe attack of pain occurred. The pain then subsided, but the patient was still unable to swallow. Examination revealed a moribund patient, and x-rays showed a large retromanubrial mass. The serologic test for syphilis was strongly positive. The course was rapidly downhill, terminating in death, Sept. 17, 1927. Autopsy revealed an aneurysm of the descending aorta encroaching upon the esophagus and here an abscess was present which communicated with the esophageal lumen and an organized clot forming a common wall of the abscess and the aneurysm.

ESOPHAGOPERICARDIAL FISTULAS

CASE 5 (B. H. No. 52602).—C. J. C., a 6-day-old female infant, was brought to the Barnes Hospital with a history of episodes of cyanosis while nursing and repeated vomiting. Four days following birth a collapse of the left upper lobe was noted. Gavage tubes had been passed prior to admission, and a barium examination had been attempted. On admission, the patient was found to be underweight. Fluoroscopic examination revealed air present in the intestinal tract plus residual barium. Esophagoscopy revealed a congenital web just above the cardia. A small Levin tube was passed into the stomach following cauterization of the congenital web. The patient seemed to progress well with the tube feedings until the age of twenty-nine days when there was recurrence of vomiting. Pneumonia developed rapidly and the infant died three days later. Post-mortem examination demonstrated a defect 1 cm. in diameter on the anterior wall of the esophagus at the level of the bifurcation of the trachea. This defect in the mucosa led into a diverticulum which extended downward and posterior to the pericardial cavity with which it communicated by means of a small opening 2 mm. in diameter. A purulent pericarditis was present. Two centimeters above the carina, a small fistula 1 mm. in diameter communicating with the esophagus was also found.

CASE 6 (B. H. No. 111650).—H. H. K., a 46-year-old white man, was first seen in February, 1944. He gave a history of excess salivation and intermittent regurgitation for eighteen years, with marked increase in severity of symptoms the past eighteen months, associated with thirty-four pounds weight loss. He had no pain but claimed he felt unable to fill his stomach. A barium x-ray examination had been done at another hospital four months previously and had revealed "a diverticulum of the esophagus just above the diaphragm." Physical examination revealed marked emaciation and dehydration. The liver edge was palpable 5 cm. below the costal margin. Esophagoscopy showed "a large diverticulum just above the cardia, which was entered easily. As a matter of fact one could hardly go any other way. This sac was filled with all kinds of meat. The mucosa was not macerated." Operation was carried out (by E. A. G.), Feb. 22, 1944, utilizing a transpleural approach. The lower esophagus was freed, revealing a diverticulum measuring 12 cm. in diameter arising on the right side about 5 cm. above the hiatus. The stomach was brought up and anastomosed to the diverticulum by an open anastomosis in two layers and reinforced with omentum. The diaphragm was then closed upon the stomach and the pleural cavity drained. The postoperative course was complicated by an empyema, which was drained, but the patient

was discharged April 16, 1944, "able to take soft food very well." He complained of some anorexia and indigestion when seen one month later.

He was readmitted Sept. 25, 1944, complaining of occasional regurgitation, pain in the left side of the chest radiating to the left shoulder for two weeks, chills and fever. Physical examination disclosed marked cachexia and "the heart sounds completely obscured by

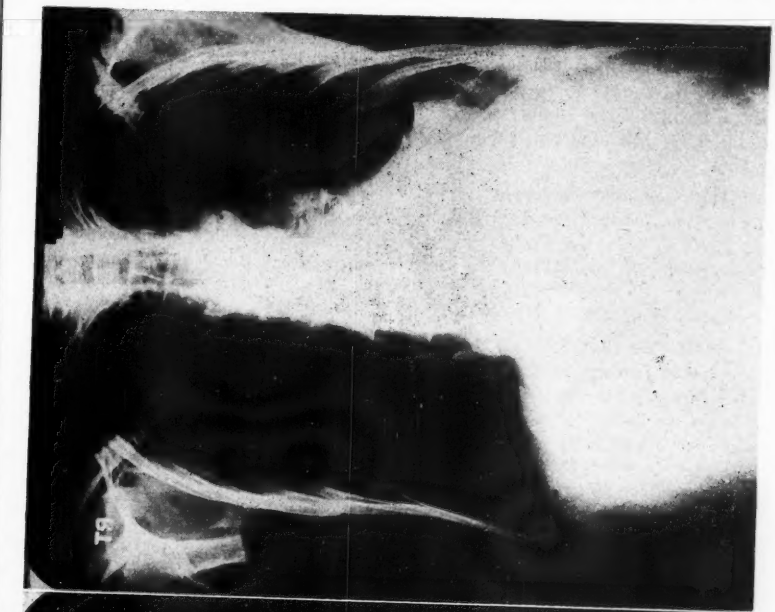

Fig. 3.

Fig. 4.

Fig. 3 (Case 6).—H. H. K. X-ray of chest following ingestion of barium. The pericardium is widely distended with air, but no fluid levels are seen. A portion of the esophageal diverticulum is demonstrated just above the left diaphragm.
Fig. 4 (Case 6).—H. H. K. X-ray taken during barium studies revealing air, fluid, and barium in the pericardial sac.

gurgling and clinking sounds of fluid swishing around in a cavity, audible without the aid of a stethoscope." There was also ankle edema and moist râles over both lower lung fields. The blood pressure was 110 mm. systolic and 80 mm. Hg diastolic. He was orthopneic, and the venous pressure was 160 mm. of water. It was noted, September 27, that his epigastric and precordial pain, which radiated to both shoulders, was considerably relieved on leaning for-

ward. The heart sounds were poorly audible on leaning forward but "in the semi-recumbent position a sticking, loud splashing sound synchronous with cardiac systole was audible." The electrocardiogram showed slightly elevated S-T segments in Leads I, II, and III as may be seen in pericarditis. A barium swallow was done and the barium seen to enter the pericardial sac on fluoroscopy. Pericardicentesis obtained air and pus, containing numerous hemolytic streptococci. A pericardiotomy was performed, Oct. 7, 1944, which revealed thickened, chronically inflamed pericardium and epicardium but the cavity itself was empty (patient was semirecumbent). Adequate drainage was instituted. The next day gentian violet ingested by mouth was seen to appear immediately through the pericardiotomy wound. Being unable to pass a Levin tube for feeding purposes, a jejunostomy was performed. The patient was progressing fairly well until Oct. 23, 1944, when he died suddenly due to a massive pulmonary embolism.

Post-mortem examination disclosed a partial stenosis of the esophagus below the diverticulum with a fistula between the diverticulum on its lateral wall going directly into the posterolateral surface of the pericardial cavity. The fistula measured 3 cm. in diameter. Microscopic sections of the tissue lining the fistula revealed chronic nonspecific inflammation.

LESIONS SECONDARY TO INSTRUMENTATION

CASE 7 (B. H. No. 94713).—J. G. H. was a 47-year-old white man, a physician, who entered the hospital in January, 1942. Following the forced passage by mouth of a bougie during a medical fraternity initiation, the patient developed a productive cough. For sixteen years prior to admission, he had coughed up food particles on occasions. He had had five severe attacks of right lower lobe pneumonia with productive cough, vomiting, fever, and epigastric pain. For the past six years, he had had to cough up fluids if he drank them with his head bent forward. Three months before entry, a fistulous tract between the esophagus and right bronchus was found on barium x-ray examination by his local physician. This was confirmed at the Barnes Hospital with the ingestion of lipiodol, and bronchograms further revealed an extensive bronchiectasis of the right lower lobe. Bronchoscopy revealed the fistula to open into the lower portion of the right main bronchus. Bronchial secretions collected from this area were tested for an analysis content and revealed 11,560 units.

A right lower lobe lobectomy was performed Jan. 21, 1942 (by E. A. G.). Extremely dense adhesions were present, most marked in the region of the posterior gutter. A very incomplete fissure was found. Due to extremely dense fibrotic and inflammatory reaction about the hilus, a mass ligation was demanded. The fistulous tract was therefore not visualized and the chest was closed with two drainage tubes to an underwater seal. A severe postoperative empyema developed. Food particles were seen to come out through the thoracotomy tube, Jan. 30, 1942, so a continuous indwelling Levin nasal tube was inserted. A thoracotomy revision was done on the seventeenth postoperative day. Despite a temporary improvement he developed severe chills and fever to over 40° C. and became extremely toxic, Feb. 13, 1942. A *Str. Viridans septicemia* was discovered. Sulfonamide therapy had to be discontinued because of leucopenia, and, despite supportive measure, the patient succumbed Feb. 28, 1942.

Post-mortem findings were (1) esophagobronchopleural fistula on the right side; (2) bronchopleural fistulas on the right side; (3) thrombosis of the splenic artery; (4) infarcts in the spleen and left kidney with abscess formation; and (5) bronchopneumonia of the left lower lobe. Detailed report of the esophageal fistula revealed that the esophagus at the level of the bifurcation of the trachea communicated directly with a small bronchus in the stump of the lower lobe of the right lung and with the right pleural cavity. The tract to this communication was lined with a definite lining membrane. On tracing this fistulous tract there was found to be a bifurcation at the hilar region of the lung, one branch of which entered a small bronchus, the other directly piercing the pleura. On microscopic section this fistula was found to be lined by stratified squamous epithelium.

ESOPHAGEAL FISTULA POSTOPERATIVE

CASE 8 (B. H. No. 118953.—T. M., a 74-year-old negro man, was admitted to the Barnes Hospital, Oct. 24, 1944, with a history of progressive dysphagia and weight loss of thirty pounds in the preceding four months. Examination disclosed cachexia, while barium and esophagoscopic studies revealed an adenocarcinoma at the esophagogastric juncture. No evidence of metastasis was found. A transpleural resection of the lower esophagus and cardia with primary anastomosis was done Nov. 6, 1944 (by E. A. G.). A thoracotomy tube was placed near the site of anastomosis. Despite intensive penicillin therapy, a definite empyema

developed on the eighth postoperative day. Gentian violet given by mouth on the eleventh day was noted to appear immediately through the thoracotomy tube. A jejunostomy was performed and all oral feedings discontinued. The patient improved slowly and by the twenty-ninth day no further evidence of fistula could be noted by oral lipiodol x-ray examination or dye tests. Progressive oral feedings were instituted and he was discharged Dec. 29, 1944. When last heard of in April, 1945, he was essentially asymptomatic with no evidence of recurrence of tumor or fistula.

DISCUSSION OF CASES

These case reports serve to emphasize certain fundamentals relative to the diagnosis and management of this complication. The history of choking paroxysms on ingestion of fluids is the most dramatic and suggestively diagnostic symptom, especially when it occurs in relation to fluids rather than solids. The latter may easily slip past a relatively small aperture. Dysphagia is a frequent precursor of the complication. The patient may describe the ability to swallow fluids without discomfort when in certain positions. Such measures may allow a long interval between the onset of the fistula and sufficient severity of symptoms to cause the patient to consult medical aid.

The diagnostic measure of greatest value consists of x-ray visualization of the fistula, preferably with iodized oil to prevent barium irritation of the lung parenchyma. Considerable difficulty may be encountered in trying to visualize these communications directly with the aid of the esophagoscope, but the instillation of methylene blue within the esophageal lumen followed by bronchoscopic examination can be a helpful measure. In cases wherein the fistula empties into the pleural cavity or through this route to the body surface, one should look for the presence of food particles, yeast cells, or the appearance of ingested dyes.

The fundamentals of therapy depend first upon the maintenance of nutrition, and second upon appropriate drainage. Many fistulas will disappear spontaneously under this management provided the etiology is noncancerous. Specific local therapy may consist of topical applications or a direct surgical approach. The question of when to resort to usual nasal tube feedings and when to resort to gastrostomy may be a vital one. It would appear that a primary trial on nasal tube nutrition is indicated in all instances if a tube can be passed. From the frequency with which coughing paroxysms may be initiated on each gastrostomy feeding, the advisability of jejunostomy is suggested as a preferable agent in some cases. This is more mandatory when the lesion is low in the esophagus or at the site of esophagogastric anastomosis. In view of the one successful result noted and four others mentioned in the literature, topical application with a strong cauterizing agent should be given a trial in esophagobronchial fistulas not secondary to malignancy. Should all other measures fail and surgery appear indicated, then jejunostomy should be instituted along with satisfactory thoracotomy drainage at the time of repair. In lesions secondary to tuberculosis, the outlook is extremely poor. In view of the secondary invading organisms, open drainage appears mandatory and this should be done anteriorly to allow subsequent thoracoplastic collapse measures as a means of fistula closure. Furthermore, in consequence of the chronicity of these lesions, gastrostomy or jejunostomy should be an early consideration.

CONCLUSIONS

1. Abnormal fistulous communications may occur from the esophagus into the bronchial tree, pleural cavity, mediastinum, pericardium, and more remote tissue spaces.

2. An esophageal fistula presents a serious hazard to the patient's life but not necessarily a hopeless one. This report adds six permanent closures to the fifteen previously recorded.

3. A review of the literature and a description of twenty-three additional cases has been given.

4. Two unusual cases of esophagopericardial fistulas are included in these reports. Another case, in which a fistula between the esophagus and the trachea occurred in relation to Hodgkin's disease, is described, and this communication was successfully treated. To our knowledge no previous similar instances have been reported.

5. The symptomatology and diagnostic methods have been described.

6. The management of an esophageal fistula demands acute interest in methods of the patient's nutrition and the employment of early adequate drainage. Some fistulas will close spontaneously under this regimen alone.

7. Direct surgical attack upon esophageal fistulas is applicable in some instances but can sometimes be obviated by endoscopic topical applications.

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LARGE ANOMALOUS VEIN ("LEFT VENA CAVA") ENCOUNTERED IN OPERATION FOR LIGATION OF PATENT DUCTUS ARTERIOSUS

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THE clinical features of this case have been presented elsewhere by Ziegler.[1] This report is concerned with a description of an unusual vein which was found during the operation for ligation of the ductus arteriosus.

CASE REPORT

The patient (B. L.) was a white female, aged 17 years. She was admitted to the Henry Ford Hospital, April 14, 1944, complaining of fever and "heart trouble." She had been ill for four months. A loud, rough systolic murmur was heard over the entire precordium with maximum intensity in the second and third interspaces at the left sternal border. There was a short diastolic murmur at the third interspace on the left side. A systolic thrill was palpable over the base of the heart. The blood pressure was 120/60 with the patient at rest; after exercise it was 120/40-10. Two blood cultures were positive for hemolytic *Staphylococcus albus*. Although the cardiac findings were not typical, the diagnosis of patent ductus arteriosus with subacute bacterial endarteritis was made. It was suggested that other congenital cardiac defects might be present. It was decided to give a course of penicillin, following which exploration of the ductus would be carried out.

The patient received penicillin for twelve days, 200,000 units being given by continuous intravenous drip for the first day, and 12,500 units intramuscularly every three hours thereafter.* She became afebrile immediately, but after ten days of penicillin treatment, the blood culture was still positive.

Operation was carried out May 8, 1944. Ethylene was given through a tightly fitting face mask. The left thorax was entered through the third interspace. The upper lobe of the lung was retracted inferiorly and medially to expose the great vessels. A large anomalous vein was seen lying on the aorta and following a course parallel to it (Fig. 1). Its diameter was only slightly less than that of the aorta. Medially, it disappeared into the areolar tissue of the mediastinum, appearing to enter the pericardium to the right of the aorta. It received at least one large branch from the neck. There was no pulsation or thrill in the vein and it collapsed with slight pressure. A prominent thrill was felt in the pulmonary artery.

The surgical significance of the vein was that a part of it lay over the groove between the aorta and the pulmonary artery, making access to the region of the ductus arteriosus very difficult. The pleura was incised along the inferior and medial aspects of the vein and a tape was passed around it in such a way that traction could be applied. The ductus was exposed after careful dissection with a small curved clamp. It was elected to do a simple ligation rather than to attempt a division of the ductus as performed by Gross.[2] A ligature of heavy braided silk was tied snugly, after which three turns of cellophane tape were placed about the ligated area and maintained in position with a second silk ligature. The use of cellophane was prompted by the report of Harrison and Chandy,[3] who used the material to produce fibrosis and gradual occlusion of the subclavian artery.

Immediately after the ligation, the thrill in the pulmonary artery became decreased in intensity. The blood pressure changed from 140/40 to 120/70. The chest was closed without further investigation of the vein. The postoperative course was uneventful. Subsequent blood cultures were negative. The patient has remained free of symptoms for one year. A loud and widely distributed systolic murmur can be heard and a systolic thrill is palpable. The diastolic murmur is absent. It is presumed that the patient has an interventricular septal defect.

From the Division of General Surgery of the Henry Ford Hospital.
Received for publication June 20, 1945.

*The penicillin was obtained through Dr. Roy D. McClure from Dr. Chester S. Keefer, Chairman of the Committee on Chemo-therapeutic and other Agents of the Committee on Medical Research.

COMMENT

A portion of the literature on congenital anomalies of the heart and great vessels has been consulted to see if a vein of this kind has been described. In her *Atlas of Congenital Cardiac Disease,* Abbott[4] presented a statistical chart of 1,000 cases of different defects. There were 105 instances of patency of the ductus and none of these patients had an associated anomaly of the large veins. Nine others had a persistent left *superior* vena cava. In Abbott's chapter on congenital cardiac disease in Osler's *Modern Medicine,*[5] there is a section on anomalies of the systemic veins entering the heart. Persistent left superior vena cava had occurred twenty-eight times in the series. There was no mention of a left *inferior* vena cava. She referred to the case of Nabarro[6] who described "a

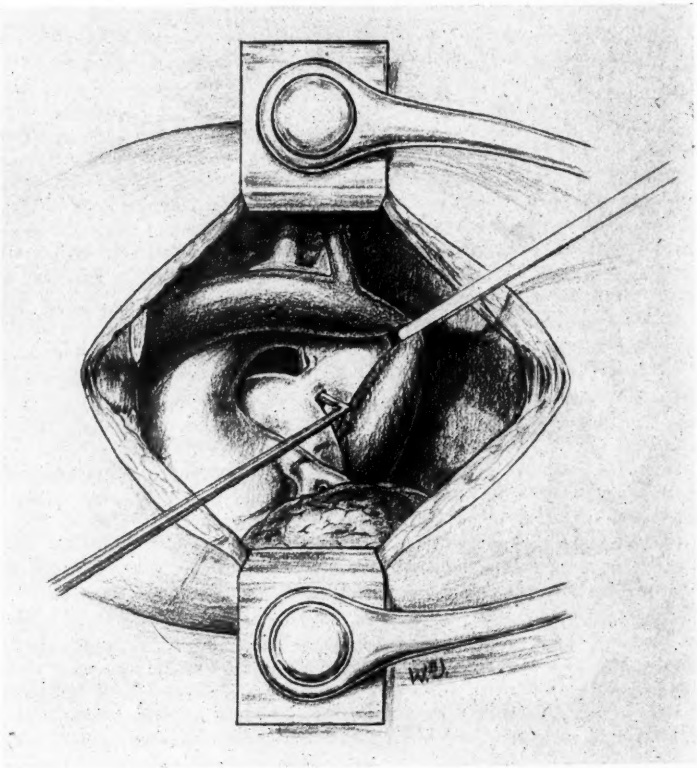

Fig. 1.—Semidiagrammatic view of findings at operation, showing the "left vena cava" being held away with the tape to expose the ductus arteriosus, which is in the curve of the aneurysm needle.

case of double superior vena cava in an infant of 3 months where the persistent left duct, smaller than the right, was joined by the left hepatic vein, which emptied with it into the coronary sinus. Here the left horn of the embryonic sinus venosus had evidently escaped obliteration." It is assumed that the term "persistent left duct" refers to the left duct of Cuvier. The present case is probably different from Nabarro's case, because the vein did not appear to follow the course of the oblique vein of Marshall to enter the coronary sinus.

Arey[7] showed diagrams of the following anomalies of the systemic veins: double superior vena cava, accessory left vena cava emptying into the left

renal vein, and double azygos with absence of the vena cava above the kidneys. He mentioned that paired or unpaired left inferior venae cavae may occur, but did not indicate a reference to a published case.

The simplest explanation for the vein in this case is to assume that it represents the left innominate and a persistence of a left cardinal vein.

It is hoped that an exact anatomic diagnosis, such as would be obtained by autopsy, will not be available for many years.

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DRAINAGE OF CAVITIES IN BILATERAL PULMONARY TUBERCULOSIS

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IN BILATERAL pulmonary tuberculosis the treatment of cavities is often the most serious problem, and it is frequently insurmountable. At times, the outlook of persons with this condition is hopeless. The degree of acute exudative tuberculosis associated with the cavities is most often the determining factor. But in many cases, the cavity alone prevents recovery, and it must be eliminated in some way.

I have treated a large number of patients with bilateral disease successfully with bilateral artificial pneumothorax. In not a few of these cases, it has been necessary to sever adhesions on one side or on both sides of the chest in order to make the collapse successful. The majority of the persons so treated have recovered eventually, and their lungs have been successfully re-expanded.

When pneumothorax cannot be used because of too extensive adhesions, it is necessary, when considering other forms of collapse, to study the patient from the standpoint of preserving adequate lung space. In treating cavities at the apex or very near the apex, bilateral extrapleural pneumonolysis and extrapleural pneumothorax has rarely been employed successfully. In exceptional cases where sufficient lung space may be saved, bilateral thoracoplasty has been employed.

However, if the cavity to be treated lies in the lower part of the upper lobe, a middle lobe, or the upper part of the lower lobe, one of these collapse

Read (by title) at the Twenty-fifth Annual Meeting of the American Association for Thoracic Surgery, Chicago, Ill., May 5 and 6, 1944.

methods is not available, because collapsing the cavity will also collapse too much useful lung. In these cases, then, I believe some type of cavity drainage must be employed.

The logic of drainage has been shown by Eloesser[1] and others. However, open drainage of tuberculous cavities has ordinarily been disastrous. I did, some ten years ago, inadvertently open a huge cavity and was so fortunate as to be able, one year later, to heal the patient by thoracoplasty; but this was unusual.

The closed method of Monaldi[2] has been used by many. In this, catheter drainage is employed. Many cavity closures have been reported, but also many patients have shown a reopening of cavities after original closure.[3] This method has been used frequently as a preliminary to thoracoplasty. In this procedure constant suction must be maintained by some mechanical apparatus.

Brunn and associates[4] reported open operative drainage and healing in a case of post-thoracoplasty cavity.

Rogers, Shipman, and Daniels[5] performed flap drainage in a number of cases of post-thoracoplasty cavity, causing healing. Acting largely on the influence of this last group of cases, I have carried out Eloesser-flap drainage in two cases of bilateral pulmonary tuberculosis which were not suitable for any form of collapse therapy. One case is of particular interest, because the patient had not only severe pulmonary disease, but also laryngeal tuberculosis, yet he has become self-supporting.

CASE REPORT

CASE 1.—A man, aged 37 years, was first seen May 7, 1938. He had advanced bilateral pulmonary tuberculosis and laryngeal tuberculosis. Neither the father nor the mother had tuberculosis and he had three brothers all in good health. His first wife died of tuberculosis seven years before. She had advanced pulmonary tuberculosis for a period of three or four years before death.

In June, 1936, he caught cold and had a persistent cough and sputum. In September, 1936, he stopped working for five weeks and he then returned to work feeling well, and weighing more than he had ever weighed before in his life. His cough disappeared and he continued to be well until November, 1937, when he again caught cold and began to cough and expectorate. About the last part of April, 1938, he had what was called "the flu," with a very high fever. He had an x-ray picture made which showed trouble in the lungs which brought him to me. He had been hoarse for about three months, and tired easily and felt weak. He had occasional nocturia.

Physical examination showed a man 70 inches tall, weighing 147½ pounds, temperature 98° F., pulse 120. His general condition was fair. Chest examination showed marked dullness throughout the right lung, amphoric breathing over the upper half, and many moist râles following cough through the upper half. The left side of the chest showed dullness to the fourth rib and the sixth spine and increased expiratory murmur in this area. The larynx showed definite redness and swelling in the left vocal cord and definite thickening of the interarytenoid space.

X-ray examination (Fig. 1) showed a large cavity at the right apex, descending as low as the sixth posterior rib. There was exudative disease below this to about the eighth posterior rib, scattered. There was density in the right costophrenic angle. On the left side there was scattered shadow as low as the eighth posterior rib with a cavity in the lung field at the level of the sixth posterior rib, calcified spots at the left hilum, and some density along the left lower bronchial tree. Hemoglobin, 90 per cent; white blood cells, 12,900; polynuclears, 74 per cent; lymphocytes, 20 per cent; monocytes, 6 per cent; sedimentation time, twenty-five minutes. Urinalysis was normal, Wassermann negative.

He was put to bed and was not allowed to talk. He improved generally, felt better, and gained weight. Examination over four months later (Fig. 2) showed that there was a definite improvement in the left lung, but the cavity in the right lung had grown larger

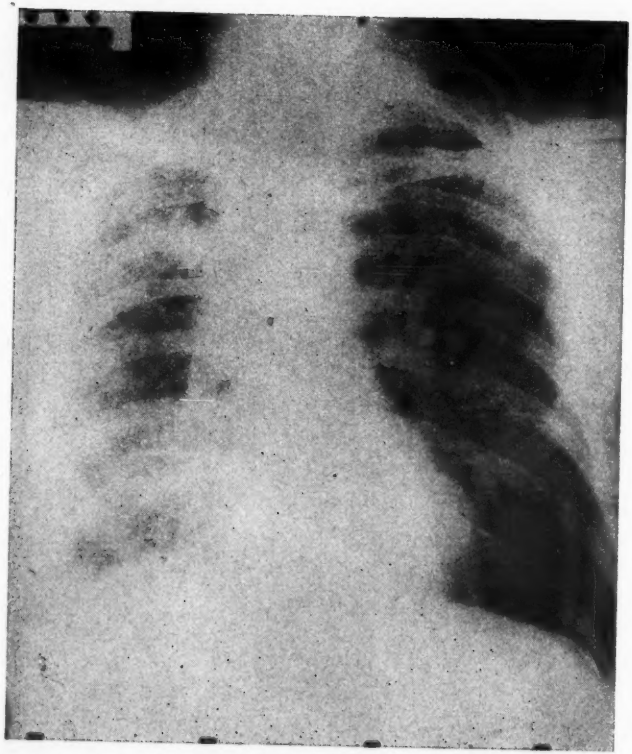

Fig. 1.—Large cavity in right upper lung, small cavity upper left lung.

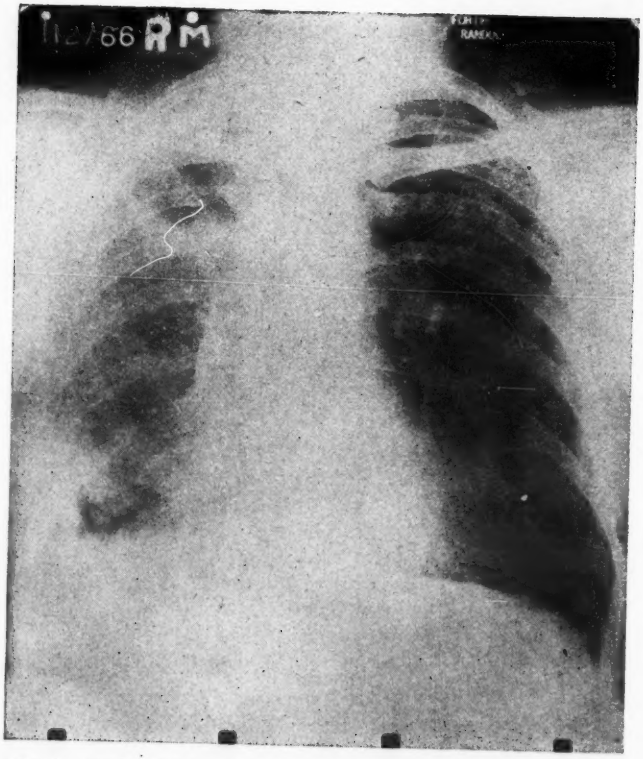

Fig. 2.—Cavity in right lung has grown larger, in left lung smaller.

and now extended as low as the seventh posterior rib. The lung field below this level showed somewhat greater shadow than previously. The right diaphragm was now obscured. The cavity in the left lung was reduced in size to about one-half.

At operation, Oct. 3, 1938, under sodium pentothal anesthesia, incision was made from the second to the fifth vertebra at the right of the spine and the ribs were exposed. The serratus posticus muscle was freed from the third, fourth, and fifth ribs and brought upward, and five inches of the third rib were resected near the spine and the extrapleural space entered. The lung was freed thoroughly extrapleurally as low as the seventh rib posteriorly and to about the sixth rib in the axilla and the third rib anteriorly. However, the lung could only be freed as low as the fourth rib along the mediastinum where the lung was very adherent. The wound was then closed with No. 1 chromic sutures and the serratus posticus was sutured over the line of closure. The muscles were then closed over this with No. 1 plain catgut and the skin with black silk.

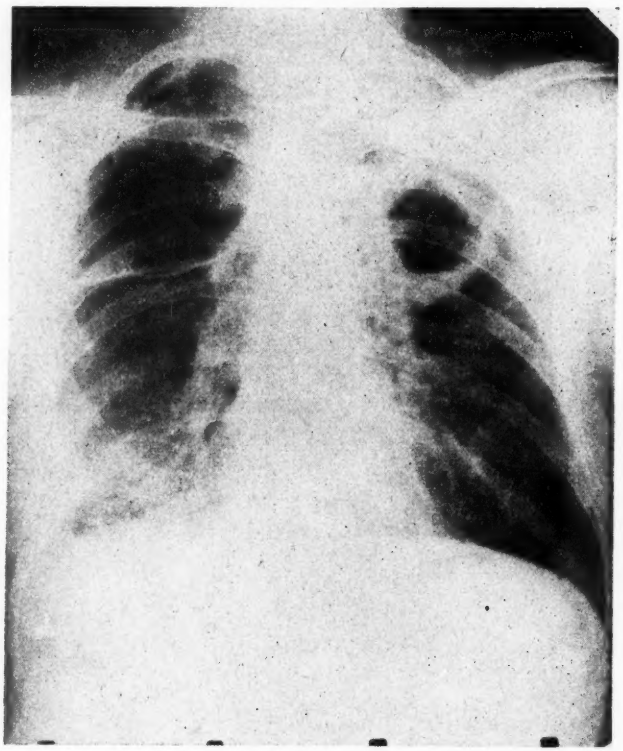

Fig. 3.—Extrapleural pneumothorax present in right; large cavity now appears in left lung.

Following surgery the patient was kept in bed as before and continued to improve. Examination, Jan. 18, 1939, showed an extrapleural pneumothorax pocket in the upper right side of the chest with cavity probably still present from the sixth to the eighth posterior rib levels. The cavity in the left lung was larger than in the previous film. Nevertheless, the patient was kept continuously in bed, pneumothorax was maintained at high pressure, and in August, 1939, we found the cavity in the right side was no longer present. There was a large extrapleural pneumothorax pocket extending below the eighth posterior rib on the right side and the collapse was quite complete along the mediastinum. The cavity in the left lung was about the same as in January and the lower right lung field showed shadow which was definitely more fibrotic in character than previously.

Since a roentgenogram, on Aug. 5, 1940, showed that the cavity in the left lung was definitely increasing in size, an operation was performed on the left side to produce an extrapleural pneumothorax similar to that on the right. The lung was stripped away

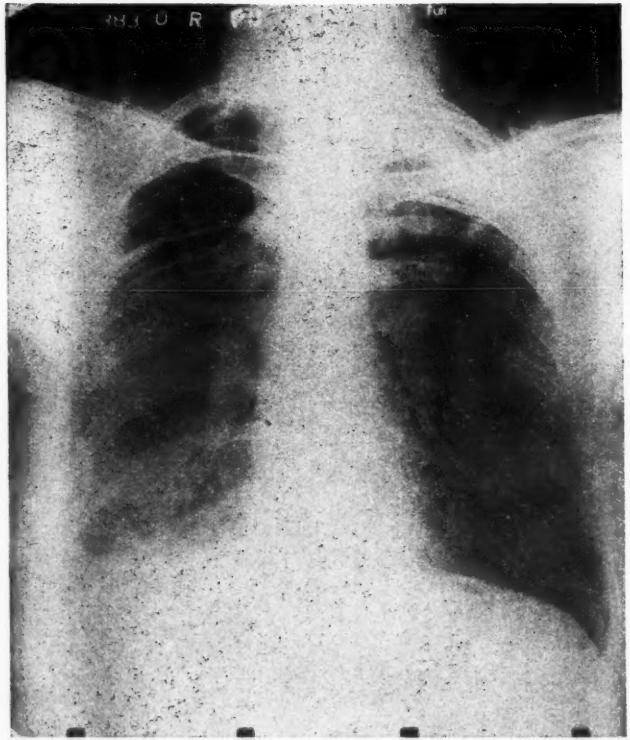

Fig. 4.—Cavity in left lung smaller; extrapleural pneumothorax still present on right.

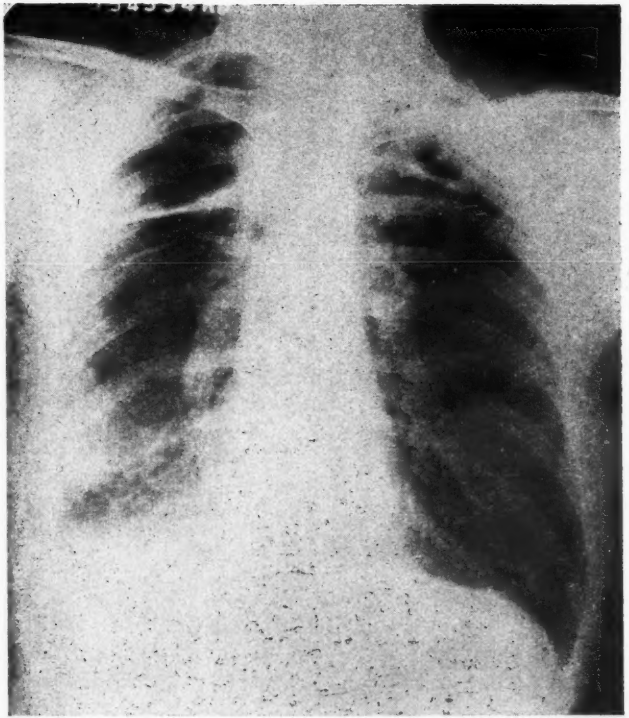

Fig. 5.—Extrapleural pneumothorax on right; active disease in chest now healed.

from the chest wall extrapleurally as low as the sixth space medially, seventh rib posteriorly, and third rib anteriorly, and as low as the fifth spine along the mediastinum. Following this, the patient's cough and sputum diminished very much and practically ceased. The general condition remained good; however, we found that fluid kept forming in this left space and was very troublesome. X-ray examination Dec. 11, 1940, showed a good pneumothorax pocket on both right and left sides, but because of the tendency of this left lung to expand, gomenol was placed in the cavity. In spite of the gomenol the extrapleural pocket continued to grow smaller and, April 5, 1941, an attempt was made to free the lung again. This failed because of dense adhesions. A very large cavity was now present on the left, running from the level of the sixth to the eighth posterior ribs (Fig. 3).

At operation, April 16, 1941, a flap was made laterally and slightly upward about three inches in length from the level of the previous entry to the pleura. The cavity was opened, the flap was sutured to the parietal pleura at the edge of the cavity and the wound was left open. X-ray examination, October, 1941 (Fig. 4), showed that the lung had entirely re-expanded and the cavity was apparently gone.

This patient was still kept strictly in bed. About this time he was allowed to start talking again and we found that his voice was quite normal. Sputum examination was negative for tubercle bacilli, Aug. 6, 1942, and remained negative thereafter on frequent examination. The extrapleural pneumothorax on the right side was continued. In 1943, this patient was allowed up from bed occasionally and in the summer of 1943 returned to work part time in a clerical capacity. In the fall of 1943 he was allowed to return to eight-hour duty, clerical work, although the extrapleural pneumothorax on the right side was still being maintained. Frequent x-ray examinations (Fig. 5) have shown the left lung to be free of disease and the lower right lung has definitely improved slowly so that we do not now find any evidence of active disease there.

CASE 2.—A woman, aged 32 years, was first seen Feb. 27, 1942. She was married but had no children. The mother had diabetes. The father, one sister, one brother, and husband were well. The mother had tuberculosis when this patient was a child and she was exposed for two or three years just prior to the age of 10. The patient had appendectomy in 1936 under gas anesthesia. Shortly after appendectomy she was found to have pulmonary tuberculosis which declared itself with hemorrhage. Shortly afterward she was started on artificial pneumothorax on the right side and made a very marked improvement. However, during 1941 she was found to have tuberculosis also in the left lung and the pneumothorax on the right side was discontinued. Late in 1941, artificial pneumothorax was started on the left side, but it was discovered that there were very marked adhesions at the left apex which prevented a cavity there from collapsing. She was then referred to me.

First examination showed that the left lung had re-expanded but there was a fairly large cavity at the left apex close to the mediastinum and extending from the level of the fourth to the sixth posterior ribs (Fig. 6). The right chest showed rather marked densities scattered in the upper and lower lung fields which appeared to be of a pleural nature. The mediastinum was drawn markedly to the right side and the trachea was curved definitely into the right chest. The function of the right lung seemed limited by scarring and contraction. It was not clear whether some active disease might not still be present. Physical examination showed a woman 63 inches tall, weighing 136½ pounds. The pulse was 100, temperature 99° F. The general condition was fair. The blood pressure was 118/80. The abdomen and extremities were negative. Hemoglobin, 65 per cent; white blood cells, 9,600; polynuclears, 76 per cent; lymphocytes, 20 per cent, monocytes, 20 per cent; eosinophiles, 2 per cent. Sedimentation time was fifteen minutes, urinalysis normal, Wassermann negative.

This patient was kept strictly in bed but had little change in symptoms or in general condition. X-ray examination, July 25, 1942, showed no essential change in the condition of the chest.

At operation, Aug. 7, 1942, an incision was made from the level of about the third vertebra to the sixth, starting about one inch to the left of the spine and continuing downward. The ribs were exposed and a section about one and one-half inches long was taken from the fourth rib and the lung was then stripped extrapleurally to the apex and along the mediastinum as low as the fifth interspace. There was so much motion in the lung as it was stripped that I decided the patient would be very short of breath if stripping were carried out for extrapleural pneumothorax. Therefore, a skin flap was created about three and one-half inches long upward from the level of the fourth space and the bottom of this was sutured to the perietal pleura at the opening. It was my idea that at a later time, after

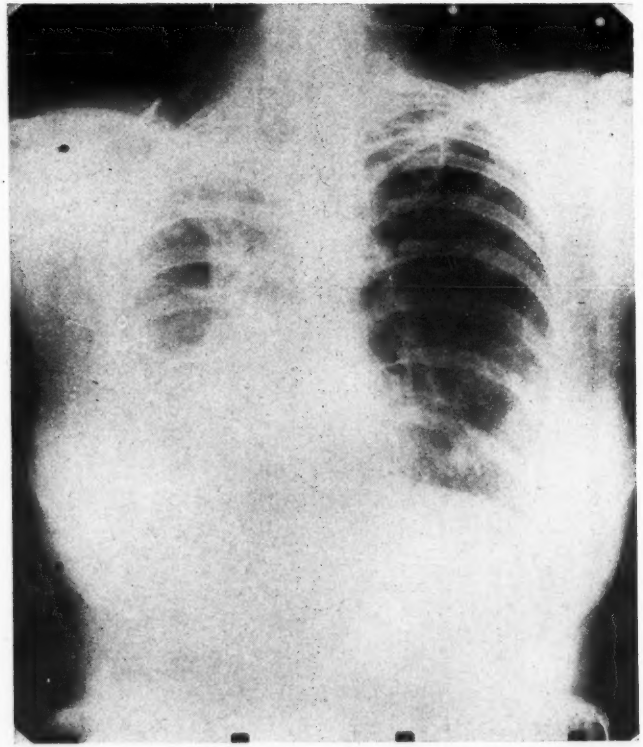

Fig. 6.—Cavity in left upper lung; right lung shows scarring and contraction from prolonged pneumothorax.

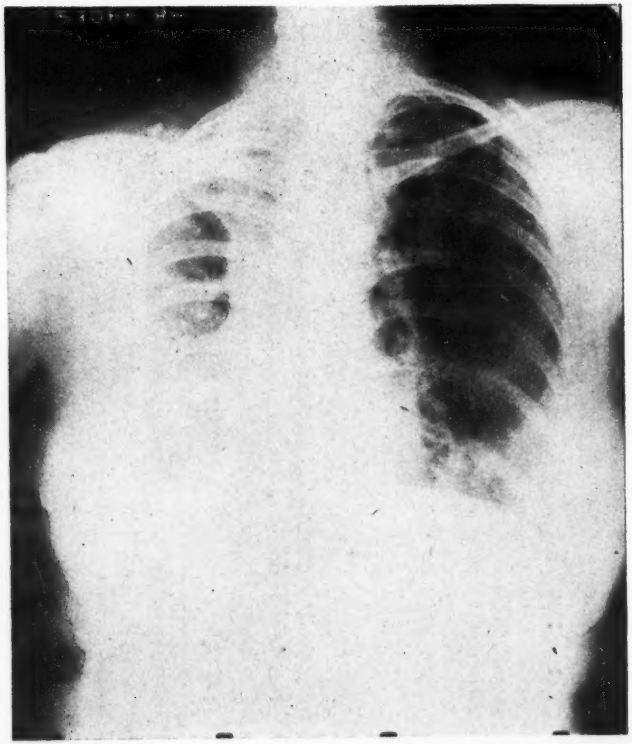

Fig. 7.—Cavity in left lung gone; right lung unchanged.

re-expansion of the lung had occurred, an opening might be made into the cavity to permit drainage through this flap. Following this procedure she was kept in bed. The lower two inches of the flap soon turned color and it was evident that the blood supply had been interrupted. This was not understandable because the flap was at least two inches wide at the upper margin of this area and two and one-half inches wide at the top. Nevertheless, this whole area became necrotic and it was necessary to wait for this to heal before proceeding with the original intention. After healing had occurred and considerable time had elapsed operation was performed on June 3, 1943. Another flap was created from the previous opening and was sutured to the lung which had re-expanded and the cavity was opened. A moderate amount of drainage occurred. The patient continued to remain in bed and the symptoms were not particularly changed. X-ray, Feb. 16, 1943, showed that the cavity was still present; however, x-ray, July 31, 1943 (Fig. 7), showed that the cavity was entirely gone and x-ray, March 3, 1944, showed the left lung without cavity and in good condition.

The field of this operation is definitely limited. In our present state of knowledge, I believe collapse measures should be employed for the closure of cavities when possible, and this procedure should be reserved for those cases of bilateral disease when collapse would be disastrous. So far it has been used only on one side of the chest where it has been possible to control the tuberculosis in the other side. I believe it may be of value for use in both sides of the chest.

It has an advantage in bilateral disease where known tension cavities exist which may not yield to collapse measures such as thoracoplasty and extrapleural pneumonolysis and pneumothorax.

It is better, in my opinion, than Monaldi drainage which introduces a foreign body into the chest. Furthermore, experience has shown that not all cavities closed by the Monaldi method remained closed. This form of drainage has the advantage that it is a simple procedure and does not require close continuous aftercare.

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A MEDIASTINAL CYST OF GASTRIC ORIGIN

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MEDIASTINAL cysts of gastric origin are relatively rare. In a recent review, Olken[1] collected twelve cases from the literature and reported one personal case. In addition, two cases have been reported by Schwarz and Williams[2] and three new cases by Ladd and Scott.[3]

In fifteen of the eighteen reported cases, the cyst was present on the right side. Mention is made of attachment of the cyst to the esophagus in eight cases. With the exception of one case reported by Schwarz and Williams in a 23-year-old man, all have been in children below the age of 4. A great majority of the patients apparently exhibited clinical manifestations.

We are reporting the case of an asymptomatic mediastinal cyst of gastric origin occurring in a 15-year-old girl.

CASE REPORT

A 15-year-old girl was referred by Dr. E. M. Kay because of a shadow which was discovered in the superior mediastinum on the left side during a routine photofluorographic school survey. Two years previously a school physician had discovered a cardiac murmur; otherwise there was nothing of significance in the past medical history.

Roentgenographic examination by Dr. S. A. Morton revealed a roughly spherical shadow about 8 cm. in diameter in the posterior mesial aspect of the left hemithorax opposite the third to sixth ribs posteriorly (Fig. 1). There was some erosion of the pedicles of the fourth, fifth, and possibly the sixth dorsal vertebra. The lesion appeared homogeneous except for a minimal amount of calcification in the periphery along the superior aspect (Fig. 2). The trachea and esophagus were not displaced. No pulsation of the tumor was demonstrated either by fluoroscopy or kymography.

On admission to Columbia Hospital, Milwaukee, Wis., July 5, 1944, the only abnormal physical finding was a harsh systolic murmur heard best over the second interspace anteriorly on the left side. The provisional diagnosis was a mediastinal tumor of neurogenic origin. The only finding not entirely congruent with this diagnosis was the calcification in the periphery of the tumor.

Thoracotomy was performed July 7. Through a posterolateral incision the fifth rib was resected, and the fourth and sixth ribs were divided posteriorly. A cystic tumor was found posterior to the parietal pleura measuring approximately 12 by 5 by 5 cm. The tumor appeared to be entirely cystic except for a nodule of firm tissue measuring 2 by 1 cm. at the inferior pole. A needle was inserted into the cyst, and moderately thick yellow, milky fluid was withdrawn. This was reported to contain cholesterin crystals and degenerated cells. The tumor was removed piecemeal after evacuation of its contents. It was densely adherent to the aorta and the vertebral bodies but apparently was not attached to the esophagus. The patient's convalescence was uneventful. The cardiac murmur was not heard after removal of the cyst.

Pathologic examination by Dr. G. H. Hansmann revealed that most of the cyst wall was approximately 1 mm. in thickness. In some areas there was calcification of the wall. The inner surface of the cyst was irregularly covered with atheromatous material. Several firm nodules were present. Microscopic examination of the sections from the wall showed

From the Department of Surgery, Marquette University School of Medicine.
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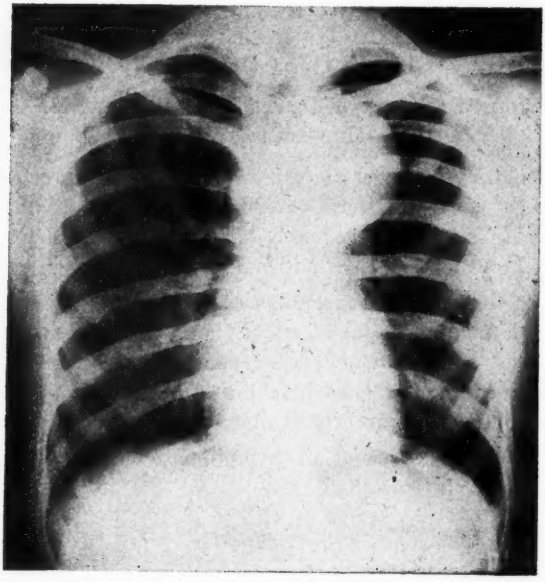

Fig. 1.—Roentgenogram of chest showing mass in superior mediastinum on left side.

Fig. 2.—Roentgenogram taken with Potter-Bucky diaphragm showing detail of tumor. Note calcification along superior aspect.

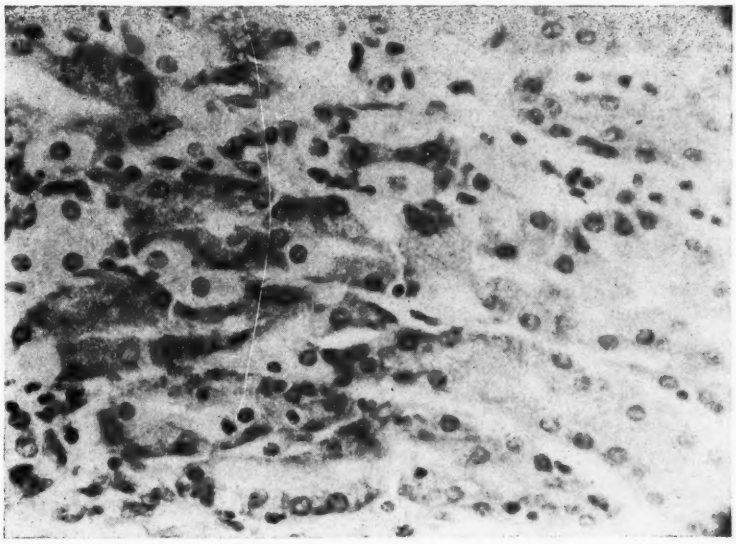

Fig. 3.—Photomicrograph demonstrating cells of gastric origin. Note both parietal and chief cells (×500).

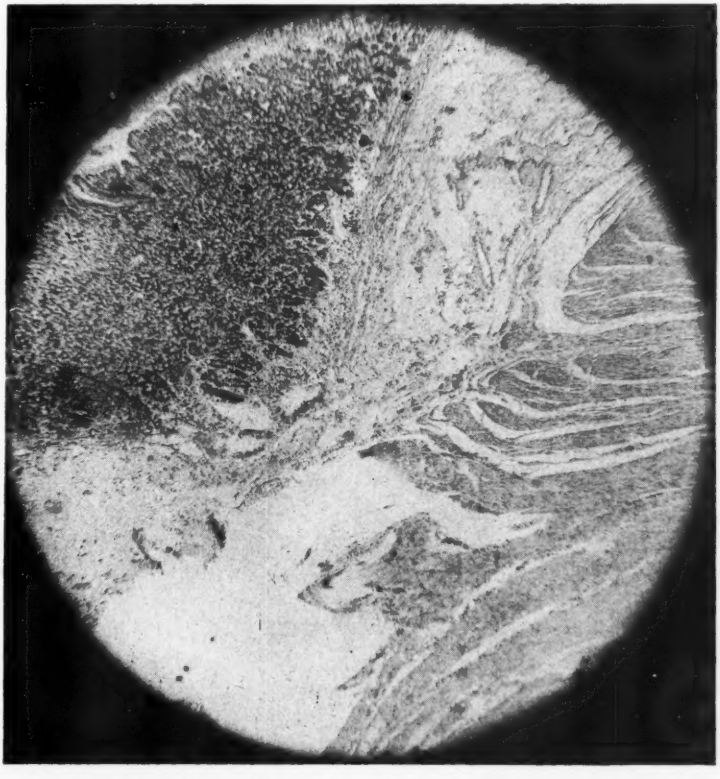

Fig. 4.—Low-power photomicrograph revealing all coats of stomach, mucosa, submucosa, and muscularis (×35).

an outer layer of fibrous tissue. In some parts of the wall there was abundant nerve tissue. Small amounts of lymphoid tissue were present, and there were areas of calcification. Bone was formed in small amounts and appeared to be derived from the fibrous tissue. Over much of the inner surface there was an irregular layer composed largely of phagocytic endothelial cells, many containing blood pigment. Cholesterin crystals and giant cells were present in some areas, and in places there was fresh hemorrhage. Sections from the nodule showed it to be covered by mucosa which structurally resembled the mucosa of the fundus of the stomach (Fig. 3). Both chief and parietal cells were present. Beneath the mucosa were bundles of smooth muscle (Fig. 4). The pathologic diagnosis was a gastric cyst of the mediastinum.

Although no chemical analysis of the fluid recovered from the cyst was made, it would appear highly unlikely that the gastric mucous membrane lining this cyst had any functional activity; otherwise the cyst would not have remained relatively small and asymptomatic over a fifteen-year period.

SUMMARY

An asymptomatic mediastinal cyst of gastric origin occurring in a 15-year-old girl is reported. With the exception of one other case, all previously reported mediastinal cysts of gastric origin have been discovered in young children.

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NEUROFIBROMA OF THE DIAPHRAGM

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PRIMARY tumors of the diaphragm have been reported in the literature as isolated case studies since the first description of such a tumor, a lipoma, by Clark[1] in 1887. Gale and Edwards[2] in 1939 reviewed the literature on this subject and presented the summaries of twelve cases of primary tumors of the diaphragm, adding one case of their own. Cases reported since that time by Ballon and Spector,[3] Ackermann,[4] Perry and Smith,[5] Soto,[6] Ryan,[7] and Hyman and Lederer[8] bring the total number of primary tumors of the diaphragm reported in the literature to seventeen. The case of Dalzell,[9] which was included by Gale and Edwards in their review of the literature, was reported as a round-cell sarcoma and probably represents a pulmonary carcinoma, therefore it is not considred in this report.

The majority of case reports were based on autopsy findings and in only seven patients was the tumor removed at operation. Of the necropsy cases there were two fibrosarcomas,[8, 10] two rhabdomyosarcomas,[5, 7] two lipomas,[1, 3] and one each of myoblastic sarcoma,[11] myosarcoma,[12] leiomyosarcoma,[12] and angiofibroma.[13]

In 1911, Gross[14] resected a large fibrosarcoma from the left diaphragm in a 30-year-old man. The patient died of recurrence seven months later. One large and four small fibromyomas were removed by Bonamy,[15] in 1912, in a 34-year-old woman, from the right diaphragm. The total weight of the tumor was 1,200 Gm. In 1913, Sauerbruch[16] removed a fibromyosarcoma of the left diaphragm in a 43-year-old woman. The patient made an uneventful recovery and no recurrence was reported. The first lipoma of the diaphragm was removed by Söderlund,[17] in 1937. The tumor was the size of a hen's egg, and the patient recovered. A somewhat larger lipoma was removed by Soto[6] from the left diaphragm of a boy of 14 years, with recovery. Ackermann[4] reported the resection of a large fibrosarcoma of the right diaphragm in a man of 27 years. The eventual outcome of this case is unknown. An endothelial sarcoma of the right diaphragm was resected by Gale and Edwards[2] in a 54-year-old man, with no evidence of recurrence nine months after the operation.

Of both the autopsy and operative specimens, eleven tumors were malignant and only six benign. The tumors reported originated in all the tissues which constitute the diaphragm, with the exception of nerve tissue. There were six myogenic sarcomas, five tumors of connective tissue origin, four lipomas, and two neoplasms arising in blood vessels. We have recently had under our observation a patient with a large neurofibroma of the right diaphragm which was removed surgically; the patient recovered. Search of the literature has failed to disclose a primary neurogenic tumor arising in the diaphragm and it is for this reason that we present the following case.

From The Departments of Research Surgery, Surgery and Medicine, The Ohio State University.

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CASE REPORT

L. Z. (No. 446874), a 42-year-old white mechanic, was admitted to the Starling-Loving University Hospital, Aug. 4, 1944, with the complaints of soreness in the joints of the extremities and swelling of the hands and feet. These symptoms had been present for one week; however, he had been aware of a backache and soreness in the right lower chest with a radiating pain extending to the umbilicus, for several months. For one year prior to admission the patient had also had a nonproductive cough, associated with occasional headaches and periods of hoarseness. There had been no hemoptysis or dyspnea. The patient had always been in good health.

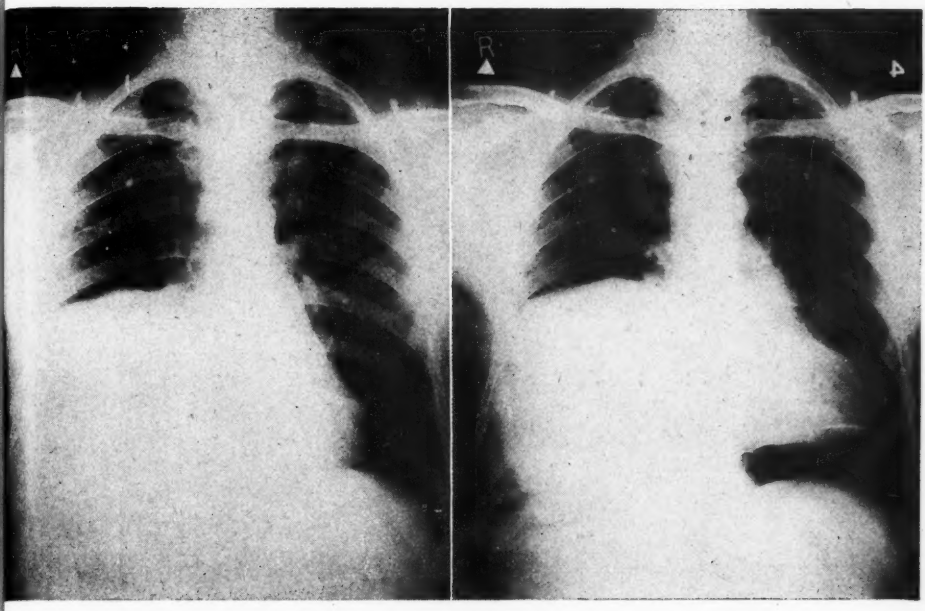
Physical examination revealed a fairly well-developed man, who did not appear to be ill. Temperature was 100° F., with a pulse of 108 and respiration of 28 per minute. The blood pressure was 110/78 mm. and was equal in both arms. The vital capacity was 1,800 c.c. There was slight injection of the mucous membranes of the nasopharynx. The chest was symmetrical; however, there was a slight lag on the right with inspiration. There was decreased tactile fremitus at the base of the right lung with relative dullness over this region, extending up to the seventh rib posteriorly, and the fourth rib anteriorly. The breath sounds were absent below this level. The heart was displaced to the left. The liver was palpated below the costal margin. There was no lymphadenopathy. The joints of the extremities were warm and tender with a normal range of motion. There was slight pitting edema of the ankles. There were no clubbing of the fingers and no cyanosis.

Laboratory examination revealed a red blood cell count of 4,560,000 with a hemoglobin of 13 Gm. The total white cell count was 14,500 with 82 per cent neutrophiles and 18 per cent lymphocytes. The sedimentation rate was 1.8 mm. per minute. The urine was normal and the examination of the feces revealed no parasites. The Kahn reaction was negative. The blood urea nitrogen was 11.2 mg. per cent and the prothrombin was 77.5 per cent of normal. The electrocardiogram was normal. The blood cultures were negative. The chest fluid did not contain bacteria.

Roentgenologic examination of the chest (Fig. 1 *A*) disclosed a normal bony framework, with the trachea in the midline and a slight shift of the lower mediastinum to the left. There was a calcified Ghon complex in the right upper lung field, while the lower right thorax was completely obliterated below the eighth rib posteriorly, and the fourth rib anteriorly. The upper part of this area of increased density was convex and somewhat irregular. Fluoroscopy revealed no abnormal pulsation; however, there was limited movement of the upper border of the mass synchronous with the left diaphragm. A right bronchogram was normal, except for upward displacement of the lower lobe bronchi and uniform filling defect of the peripheral portion of these bronchi. There was no evidence of bronchial dilatation.

Hospital course: A right thoracentesis was done with the removal of 50 c.c. of a clear straw-colored fluid. A needle puncture of the mass was performed, and a small amount of bloody fluid removed, which on supravital staining was found to be blood containing an occasional cell somewhat suggestive, but not diagnostic, of malignancy. Following right pneumothorax and pneumoperitoneum, x-ray films (Fig. 1 *B* and *C*) visualized a large tumor continuous with the diaphragm and the right lower lobe of the lung. A preoperative diagnosis of an intrathoracic tumor was made, possibly a bronchogenic carcinoma or teratoma, and an exploratory thoracotomy was performed Aug. 24, 1944.

The operation was performed under cyclopropane-ether-oxygen intratracheal anesthesia. Using a right posterolateral incision, the thorax was entered by resecting the eighth rib from its angle to the costocartilaginous junction. The ninth rib was sectioned at its angle. The seventh, eighth, and ninth intercostal nerves were crushed. The parietal pleura was normal. Exploration revealed a hard, irregular, somewhat lobulated mass filling the lower half of the right thorax, and attached to the diaphragm by a pedicle approximately 8 cm. in diameter. The tumor was firmly adherent to the diaphragmatic surface of the lower lobe of the lung. The lower and middle lobes were somewhat compressed, but were not atelectatic. There was no mediastinal mass or hilar lymph node enlargement. Large veins and arteries were crossing into the tumor through the pedicle. The pedicle of the tumor was incised at its base, and the central portion of the diaphragm was removed with the tumor. The pedicle was extremely vascular and suture ligatures were used to secure hemostasis. Gauze dissection was used to free the tumor from the lower lobe. The phrenic nerve was crushed as it crossed the pericardium. The defect in the diaphragm was repaired without difficulty by using a row of interrupted mattress sutures of chronic No. 1 catgut. The defect in the pleura of the lower lobe, where the tumor had been attached, was repaired with interrupted sutures of No. 00 chromic catgut. The chest was closed using chromic catgut for the muscles and silk



A. *B.*

C.

Fig. 1.—*A*, Roentgenogram of chest on admission, showing complete obliteration of the right lower thorax by the tumor. *B*, Right pneumothorax and pneumoperitoneum outlining the diaphragm and the lateral and lower border of the tumor. *C*, Right oblique view, showing large size of tumor.

for the skin. A No. 34 Pezzer catheter was inserted in the ninth interspace in the mid-axillary line for drainage. The patient was given 300 c.c. of whole blood during the operation.

Postoperative course: The patient made an uneventful recovery and had very little postoperative pain. The drain had been connected to a water trap and was removed in forty-eight hours, the drainage at that time being serous, and x-ray showing complete re-expansion

A.

B.

Fig. 2.—*A*, Photograph of tumor, showing the pedicle and area of attachment to the diaphragm. *B*, Cut section of tumor; dark area is site of needle puncture and subsequent hemorrhage.

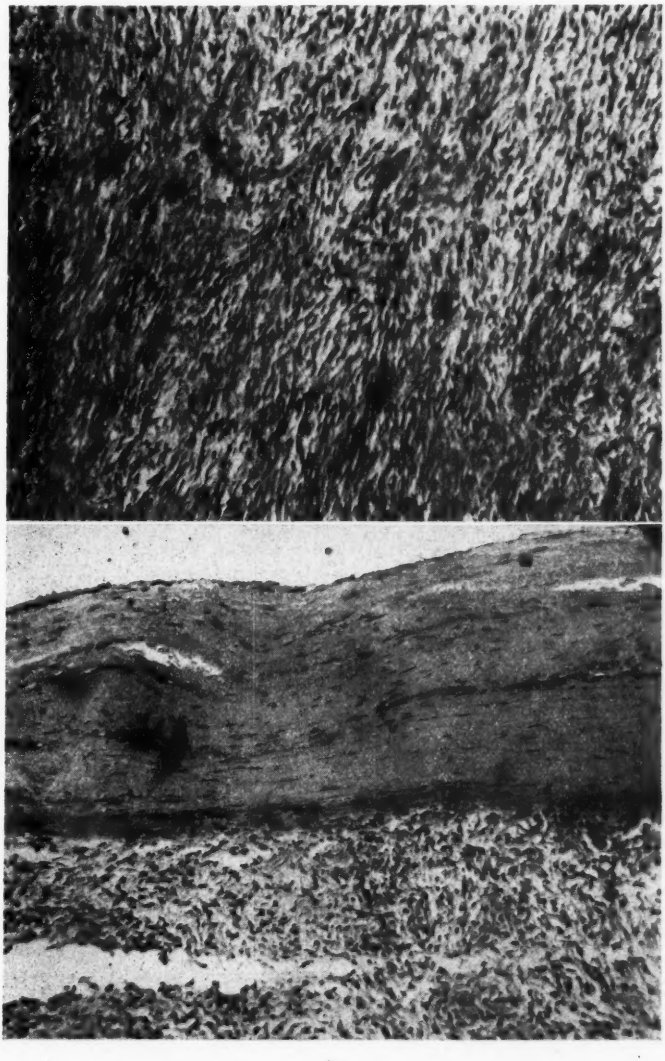
of the right lung. The patient was out of bed on the fifth postoperative day, and discharged on the twelfth. On the day of discharge his vital capacity was 2,200 c.c. He returned to work in six weeks, having gained about twenty pounds.

A roentgenogram (Fig. 4), taken two months postoperatively, showed an elevated right diaphragm with some retained lipiodol in the right lung. The patient was last seen six

months postoperatively, at which time he had no complaints. The diaphragm was functioning and the vital capacity was 2,900 c.c.

The pathologic examination was made by Dr. Charles Chesner. The gross specimen as removed weighed 1,800 Gm. It was somewhat ovoid, and lobulated at its upper surface (Fig. 2 *A*). It was completely covered by a dense, adherent capsule which contained large blood vessels. The lower diaphragmatic portion contained strands of muscle arranged in a radial fashion at the pedicle. On section (Fig. 2 *B*) the tumor was whitish, homogeneous, and had an area of recent hemorrhage along the right border. There was no evidence of cystic degeneration.

A.



B.

Fig. 3.—*A*, Photomicrograph of section from tumor. *B*, Section of tumor and capsule.

Sections (Fig. 3 *A*) showed interlacing whorls of cells and fibers. The nuclei were fairly regular in size, shape, and tinctorial properties. Most of the nuclei were enlarged or slightly spindle shaped. The more spindle-shaped nuclei were rather vesicular in character, while the elongated ones had a somewhat darker chromatin material. The stroma was abun-

dant, compact, and in places showed many delicate wavy fibrils. The whorled arrangement of cells and fibers was also conspicuous. Alternating with the more compact areas, there were small foci showing a looser arrangement of the intercellular material. The blood vessels were sparsely distributed throughout the tumor. A dense, fairly vascular, but acellular connective tissue capsule was present at one edge of the section (Fig. 3 *B*). The histopathologic appearance was that of a neurofibroma.

Fig. 4.—Roentgenogram of chest two months after removal of the tumor. The right diaphragm is elevated and there is retained lipiodol in the right lung.

DISCUSSION

The presenting symptoms in this patient were due mainly to pulmonary osteoarthropathy, rather than to interference with the patient's vital capacity because of the large size of the intrathoracic tumor. No clubbing of the fingers was present; however, the pain in the extremities disappeared completely following the removal of the tumor. The patient had not been aware of dyspnea, even though there was a marked decrease in the vital capacity. The neoplasm filled the entire right lower thorax, with pressure symptoms of backache and occasional pain along the distribution of the tenth intercostal nerve. The neurofibroma apparently arose in the diaphragmatic portion of the right phrenic nerve, since the central portion of the diaphragm is innervated by this nerve alone. However, the diaphragm was not paralyzed and the patient did not complain of shoulder pain, a frequent indication of phrenic nerve involvement. The phrenic nerve was visualized during the operation as it crossed the pericardium. However, no attempt was made to dissect its lower branches. Following crushing of the phrenic nerve, the diaphragm was paralyzed and remained so for six months, when its function returned.

The majority of primary tumors of the diaphragm reported in the literature was malignant. It is evident, therefore, that all such primary tumors should be removed once a diagnosis has been made or is suspected. The preoperative diagnosis may frequently be difficult, or impossible, even though such diagnostic procedures as pneumothorax with thoracoscopy, pneumoperitoneum

and x-ray studies, bronchoscopy and bronchography have been used. An exploratory thoracotomy in questionable cases is indicated, since the risk involved is minimal. The neurofibroma in our patient did not show any evidence of malignancy. The capsule was intact; however, there was definite lobulation of the upper portion of the tumor. Kent and his associates,[18] reporting from Graham's Clinic, have recently pointed out that 40 per cent of all neurogenic intrathoracic neoplasms showed evidence of malignant changes and they advocate early removal of all such tumors.

SUMMARY

A case of a primary neurofibroma of the right diaphragm is presented. Surgical removal of the tumor was followed by complete recovery of the patient.

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Announcement

Due to conditions beyond the control of the editors and publishers, several issues of the JOURNAL are printed on an inferior grade of paper. Just as soon as the standard good grade of paper is available, its use will be resumed.

SELF-RETAINING SCAPULA RETRACTOR FOR THORACOPLASTY

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BRANTIGAN and Aycock,* in 1942, advocated the use of the Sauerbruch or Tuffier rib spreader as a self-retaining scapular retractor for upper rib thoracoplasties. One blade was placed against the upper surface of the rib below the last one to be resected and the other blade against the muscle-covered tip of the scapula. This method of retraction resulted in excellent exposure of the upper thorax; however, certain difficulties were encountered. Brantigan and Aycock suggested that the blades of the Tuffier retractor be serrated to prevent slipping of the blade along the edge of the scapula. However, these shallow serrations, in our experience, did not prevent slipping of the blade along the scapular edge and it was necessary for an assistant to support the blade during the thoracoplasty. Also, on several occasions the long blade of the Tuffier retractor, set against the upper edge of a rib, penetrated the parietal pleura with the production of a pneumothorax.

To eliminate these shortcomings of the Tuffier retractor the stationary blade was shortened by 1 cm., while the sliding blade was perforated in three places, and steel pegs measuring 1 cm. in length were screwed along the outer surface of the blade (Fig. 1). These pegs were long enough to penetrate the muscles of the scapula to the bone and prevent slipping of the retractor. This

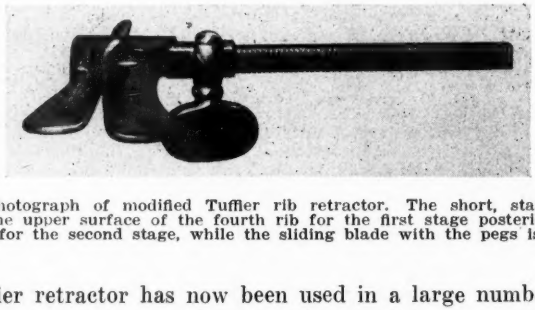

Fig. 1.—Photograph of modified Tuffier rib retractor. The short, stationary blade is placed against the upper surface of the fourth rib for the first stage posterior thoracoplasty, or the sixth rib for the second stage, while the sliding blade with the pegs is set against the scapula.

modified Tuffier retractor has now been used in a large number of patients without any postoperative complications that could in any way be attributed to the use of this retractor. The exposure of the upper four to five ribs has been excellent, in both right and left thoracoplasties, and resection of the first and second ribs has been made safer, due to the excellent visualization of the operative field.

SUMMARY

By shortening one blade of the Tuffier retractor and inserting pegs along the outer surface of the second blade, this retractor can be used as a self-retaining scapula retractor. The advantage of this modification is that no assistant is required to hold the retractor and there is no danger of perforating the parietal pleura during thoracoplasty.

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*Brantigan, O. C., and Aycock, T. B.: Self-retaining Scapula Retracting for Thoracoplasty Without Use of a Special Instrument, J. THORACIC SURG. 12: 394-396, 1943.